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# THE PRACTICE AND TEACHING OF SURGERY IN VETERANS ADMINISTRATION HOSPITALS*

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THE present Department of Medicine and Surgery in the Veterans Administration is to a considerable extent an achievement of a late member of this society, Dr. Elliott Carr Cutler. He was among the first to insist that if the Veterans Administration hospitals were under the aegis of the Universities, the highest standards of care would be assured for veterans and at the same time badly needed facilities for medical and surgical training would be provided for the many doctors whose education had been interrupted by the war. His unselfish devotion to the cause of the veteran, particularly the veteran doctor, and his lifelong interest in medical education led him to play a prominent role in establishing the aims and principles of a reorganized medical department in the Veterans Administration. It was his last great contribution to surgical progress. How effectively the ideals of Dr. Cutler have been realized is the substance of this report. Although it covers only Branch No. 1 (the New England area), it is representative in a general way of the entire country.

An account of the organization of the Veterans Administration is pertinent to the discussion. The Department of Medicine and Surgery is but one of several departments under the authority of the Administrator of Veterans Affairs in Washington. For purposes of administration the country is divided into 13 branches corresponding to the civil-service districts, each of which is under the authority of a deputy administrator and in each of which the Department of Medicine and Surgery and its representatives constitute only a comparatively small segment. From this point of view administrative problems that at the level of a hospital seem of the greatest importance may pale into insignificance by comparison with issues that confront the Veterans Administration as a whole. An appreciation of this fact is essential if one is to deal sympathetically and successfully with members of the Veterans Administration whose positions are primarily administrative.

Figure 1 depicts the organization of the Medical Department only as it applies to surgery. A similar schema could be drawn for medicine, tuberculosis or neuropsychiatry. In the interests of clarity the relations between the Department of Medicine and Surgery and the rest of the Veterans Administration have been omitted. At the top is the Chief Medical Director in Washington. Dr. Paul Magnuson has succeeded General Hawley in this important post. Under him is the Central Office Administration with various subdivisions, which are not shown but one of which is a professional services division composed of full-time men who act in an administrative capacity and a group of part-time consultants — the "national consultants," who advise the Chief Medical Director in professional matters in their respective fields. For each of the 13 regional districts there is a branch office administration headed by the branch medical director. Under him are the managers of the various hospitals. In this Branch, No. 1, there are now six hospitals, two of which — Togus, Maine, and Providence, Rhode Island — have no affiliation with

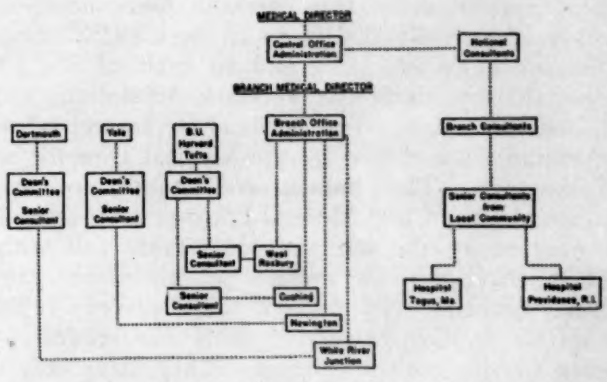


FIGURE 1. *Organization of a Medical Department as It Applies to Surgery.*

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medical schools. The other four are associated with medical schools: Newington, Connecticut, under Yale, White River Junction, Vermont, under Dartmouth, and Cushing and West Roxbury under a joint committee from Boston University, Harvard and Tufts.

A most praiseworthy feature is the comparative autonomy of the medical schools. They have no contract with the United States Government, but they have complete responsibility for all professional appointments in the affiliated hospitals. Consultants and attending surgeons and physicians are nominated by the dean's committee. The appointment of the full-time professional chiefs is authorized by the Veterans Administration only after approval by the dean's committee, the Branch Medical Director and the Hospital Manager. Residents are selected by the chiefs of services with the approval of the dean's committee. Furthermore the medical schools through the dean's committee and the professional staffs are authorized to supervise the teaching programs and to direct the expenditure of research funds. Thus, without asking anything in return the Government has given the professional supervision of the affiliated hospitals to the medical schools.

In addition to the consultants appointed by the medical schools there are, as mentioned above, those appointed by the Veterans Administration to act as professional advisers to the Chief Medical Director in Washington and to each of the 13 regional divisions of the Veterans Administration, the branch offices. The national or central office consultants are chosen by the Medical Director in Washington. The branch consultants are appointed by the Chief Medical Director after recommendation by the national consultants and with the approval of all the medical schools in that particular branch. They make detailed surveys of all hospitals in their respective fields and render reports to the branch director. They have direct professional supervision of the nonaffiliated hospitals. However, lest they interfere with the dean's committees they have no professional authority over the affiliated hospitals. As there is no established channel for direct communication between the branch consultants and the dean's committees occasional misunderstandings have arisen and in some areas it has been suggested that the former restrict their activities to the nonaffiliated hospitals.

Actually, the branch consultants, by visiting all the hospitals and maintaining a close contact with the administration, should acquire a valuable breadth of view and be in a position to clarify many problems that appear to be administrative but affect the care of patients. A recognized channel of communication between the dean's committee and the branch consultants would lead to a better integration of the consultant program. The simple expedient of having the senior consultants ipso facto members of the respective dean's committees would avoid an unnecessary link in the chain and would provide a ready means of contact between the branch consultant and the dean's committee. In hospitals where the senior consultant in surgery is a member of the dean's committee (Yale and Dartmouth — Fig. 1) such a contact has naturally developed and has been mutually beneficial.

An immediate result of the affiliation of the Veterans Administration hospitals with the medical schools was an extraordinary improvement in the caliber of the professional personnel. In 1945 there was hardly a qualified surgeon in the New England Branch. There were none recognized as such by the American Board. Now all the full-time chiefs of surgery in the affiliated hospitals are well trained men with distinguished records of individual accomplishments. Assisted by able consulting and attending staffs, they have established surgical services comparable to those in the best university hospitals.

The part of the hospital consulting and attending surgeons is a vital one. The consultants are men with established reputations as surgeons and teachers. They supervise the teaching, act in an advisory capacity and occasionally carry out surgical procedures of an unusual or formidable nature. In a word, they bring the medical school to the hospital. The attending surgeons, in general, are a younger group. Free from administrative responsibilities, they do the bulk of the daily bedside and operating-room instruction but do very little surgery themselves. It may be well to emphasize that the attending surgeon cannot be a man of limited experience hoping to broaden himself by operating in the Veterans Administration hospitals. He must be an accomplished surgeon, capable and willing to teach. His role is a comparatively selfless one.

The consulting and attending surgeons receive a per-diem allowance of $50 and $25, respectively, for their services. Since each visit consumes the better part of a day in ward rounds, at clinics or in the operating room the financial return cannot compare with that in private practice. Moreover, in assaying the cost of the consultant program to the Government one must remember that prior to the new regime a consultant was paid for his services by the operation. He might receive as much as $300 for a major procedure Now he conducts rounds, gives a clinic and assists at or performs the same operation for $50 or $25.

The accomplishments of the reorganized Department of Medicine and Surgery are best measured by improvements in the standards of care offered to the veterans. A detailed comparison between the old and the new would serve no useful purpose and is hardly possible since the older records are not adequate for analysis. The figures from one of the hospitals (Table 1) are indicative of the general trend showing a large increase in the volume

of clinical material, a reduction in the number of deaths and a gratifying increase in the percentage of autopsies performed.

The record at present speaks for itself, and since nothing appeals to the surgical mind more than a set of figures, a broad indication of the type of surgery now being done in the affiliated hospitals is presented in Table 2. The data include all operations performed for gastric or duodenal ulcer in the last two years in this branch. Since patients are not usually admitted to these hospitals unless their symptoms indicate failure to respond to therapy, the number of cases in which surgery was done is conservative. Time does not permit a detailed breakdown of the material, but the figures for gastric resection include 2 for gastrojejunocolic fistula and 8 for acute massive hemorrhage. The mortality for acute perforation is exceptionally low and the over-all mortality is most gratifying.

TABLE 1. *Comparative Statistics in a Veterans Administration Hospital.*

| DATUM | 1945 | 1947 |
|---|---|---|
| Admissions | 3852 | 5699 |
| Deaths | 201 | 150 |
| Autopsies | 42 (20%) | 110 (73%) |

TABLE 2. *Treatment of 1369 Patients* with Peptic Ulcer Admitted to Veterans Administration Hospitals (Branch No. 1) from July 1, 1946, to July 1, 1948.*

| TREATMENT | NO. OF PROCEDURES | NO. OF DEATHS | MORTALITY % |
|---|---|---|---|
| Gastric resection | 226 | 7 | 3.0 |
| Vagotomies | 55 | 0 | |
| Plication of perforations | 57 | 1 | 1.7 |
| Posterior gastroenterostomy | 5 | 0 | |
| Totals | 343 | 8 | |
| Average | | | 2.3 |

*Of these, 316 (22 per cent) were operated on.

An interest in research is characteristic of good hospitals and to a certain extent is a reflection of high standards. It is significant that two papers from Veterans Administration hospitals were presented at this meeting yesterday. To date, five papers have been published from the surgical services of the four affiliated hospitals. An additional nineteen papers covering a wide range of topics are in process of publication. Recently funds have become available for laboratory studies and special projects so that the future should see a considerable volume of valuable research done in these institutions.

From the foregoing, it is obvious that the surgical staffs and standards of the affiliated Veterans Administration hospitals are excellent for training residents if the variety and volume of clinical material are adequate. There is an unusual number of rare and interesting cases, a steady flow of the simpler types of surgery and an abundance of gastric and vascular work. The amount and type of material available in general surgery is indicated in Table 3, which lists the operations performed by the chief residents in one month in two different hospitals. Similar data could be produced for the subspecialties of urology and orthopedics, which are well represented in the majority of the Veterans Administration hospitals. Finally, there is ample material for training in roentgenology and pathology. Unfortunately, however, the restriction of the clinical material largely to one sex and certain age groups makes it essential for each resident to obtain a part of his training elsewhere. Despite this drawback, an increasingly larger number of able young men are seeking residencies in these hospitals and a means of providing them with a balanced training through affiliation of the Veterans Administration hospitals with civilian institutions is highly desirable. The Dartmouth Dean's Committee has taken the lead in this respect by establishing a unified residency between the Hitchcock Hospital and the Veterans Administration Hospital at White River Junction. All their residents are now assured a three-year graded service, which includes work at both hospitals. It is urgently hoped that other institutions will see fit to do likewise.

TABLE 3. *Operations Performed by Chief Residents in a One-Month Period in Two Veterans Administration Hospitals.*

| HOSPITAL NO. 1 OPERATIONS | NO. | HOSPITAL NO. 2 OPERATIONS | NO. |
|---|---|---|---|
| Gastrectomy | 2 | Gastrectomy | 4 |
| Colectomy | 1 | Colectomy | 1 |
| Colostomy | 1 | Colostomy | 1 |
| Cholecystectomy | 1 | Cholecystectomy | 2 |
| Thyroidectomy | 2 | Lobectomy | 2 |
| Appendectomy | 4 | Appendectomy | 2 |
| Herniorrhaphy | 12 | Herniorrhaphy | 3 |
| Lumbar sympathectomy | 1 | Miscellaneous major procedures | 10 |
| Lumbodorsal sympathectomy | 1 | Miscellaneous minor procedures | 16 |
| Miscellaneous major procedures | 8 | | |
| Miscellaneous minor procedures | 25 | | |
| Totals | 58 | | 41 |

The large volume of excellent teaching material in the Veterans Administration hospitals makes them especially suitable for affiliation with well staffed community hospitals that may not have large ward services. It is sincerely hoped that under the supervision of the universities affiliations can be established between outstanding community hospitals and the Veterans Administration hospitals that are not, at present, associated with medical schools. Unless this can be worked out, the future of the nonaffiliated Veterans Administration hospital is an unhappy one.

The wisdom of linking the Veterans Administration hospital to the medical schools is shown by the present plight of the nonaffiliated hospital at Togus, Maine. The clinical material in this hospital is comparable to that in any of the affiliated hospitals. The full-time Chief of Surgery is well trained, competent and conscientious, and there are a number of able consultants who visit the hospital regularly. A well conceived program of lectures, ward rounds and clinics is established, but because the institution is not a dean's committee hospital it is not authorized to appoint interns or residents. Consequently, the members of the regular full-time staff are overwhelmed in providing the elementary details of good care. Naturally, it is difficult to keep an able staff in such circumstances. The present chief of surgery remains because it is hoped that sooner or later a residency-training program can be instituted. If he should leave, it would be difficult to persuade a well trained and competent surgeon familiar with the situation to succeed him.

The future of the hospital at Togus and other hospitals to be set up in this branch is dependent upon an affiliation with one or more of the universities. Geographic proximity, although desirable, would not be essential. The moment a hospital is authorized for resident training, defects in the full-time staffs can be corrected because well trained men are willing to take positions in dean's committee hospitals. An able resident staff could then be appointed, and in a very short time the atmosphere and standards of these hospitals would compare with those of the other affiliated hospitals.

If one excludes the nonaffiliated hospitals from the discussion, it is evident that the reorganized department of medicine and surgery in the Veterans Administration has fulfilled expectations. It has attracted distinguished staffs, offers excellent opportunities for training and research, and provides the veteran with the highest quality of care. It deserves a continued and expanding support from the universities and the medical profession.

On the other hand, it would be a mistake to conclude that the notable achievements of the Veterans Administration hospitals are examples on a small scale of what a system of socialized medicine could do for the country as a whole. The record of the Department of Medicine and Surgery has been made despite the fact that it is a governmental system and not because of it. From the very beginning of the new regime, General Paul R. Hawley with the approval of General Bradley insisted that the "red tape" of administrative details be reduced to a minimum and that nothing should hamper the professional personnel in their work. Despite the fact that this policy, vigorously supported by Mr. Gray and Dr. Magnuson, has permeated the entire organization there is a constant struggle between the professional personnel and the Frankenstein of any governmental system.

Thus, elaborate checks and safeguards must control the spending of money that belongs to the people of the United States. A new budget must be approved each year by Congress and shared with other departments. It may be exceeded before the end of the fiscal year and suddenly precipitate reductions in personnel and travel money. Although doctors and nurses are not affected, the unique rules and regulations of civil-service employees may shift key technicians or secretaries to other positions and replace them with less suitable people. Moreover, someone must be accountable for the property of the United States Government, and despite every effort to minimize it some of this responsibility falls on the shoulders of the professional staff. The financial department requires consultants and attendants to sign in and out daily and cannot conceive of a doctor visiting a hospital for other than personal remuneration. Personnel ceilings, as well as a forty-hour week for each employee, place a serious limitation on the work that can be done in the operating room. There are well intended building regulations, which if literally interpreted may create the most preposterous situations. In one hospital the use of a badly needed autoclave was delayed for more than a year for apparent lack of authority to make a hole through a wall.

Since these are Government hospitals it is essential for the managers to have more authority than the director of a civilian hospital. They are in a sense the director and the hospital trustees all in one. Fortunately, the hospital managers in this branch have been well chosen, and excellent relations between them and their service chiefs have developed. But there are potentialities for discord, and should a manager prove to be dictatorial and unsympathetic with the aims of the professional services, he could make the lives of his service chiefs miserable. Moreover, it would take time to untangle such a situation. Furthermore, both managers and professional chiefs are subject to the well intended influence of Congressmen and service organizations who with the best of intentions may unwittingly interfere with the care of patients. Worst of all, even the consultants, because there is a flavor of governmental authority in their suggestions, at times may be more confusing than helpful.

These are not criticisms of the Veterans Administration. These are facts that, so far as it is humanly possible, the medical director of the Veterans Administration and his department chiefs have endeavored to eliminate. In this connection it can be stated parenthetically that if the Department of Medicine and Surgery had its own budget under the direction of the Chief Medical Director, a great many of the administrative difficulties that now beset the hospitals could be eliminated. But the best of governmental systems is hard to control. It requires constant and determined resistance,

and it takes a strong, patient and selfless man to hold a permanent position as chief of surgery in the Veterans Administration. As long as the organization is comparatively small and administrators like General Hawley and Dr. Magnuson remain at the top and as long as the universities continue their support, it is hoped that enough able men will be found for these positions to assure the high standards of care that Elliott Cutler envisioned. To expand this system to all the people would be disastrous. The machine would control the men, and the care of patients would be in the grip of an automaton.

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## ARACHNIDISM*

### Effect of Calcium Gluconate in Six Cases

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ARACHNIDISM, the syndrome following the bite of a black-widow spider, is a definite clinical entity in the field of general medicine. The belief that the bite of the spider *Latrodectus mactans*, present in all but seven states in America, is poisonous for man has been recognized for centuries.[1] Before Bogen's[2] review of the literature in 1926 there was much skepticism attached to the fact that such a small creature could, by its bite, produce terrifying generalized symptoms in man. He reported 380 cases with 17 deaths in eighteen states. It remained for Blair,[3] in 1933, to dispel all doubt concerning the seriousness of this arachnid menace to man when he described the clinical syndrome that followed the bite of a female *L. mactans* after application of the spider to his own finger.

The distribution of *L. mactans* in the United States is wide; this spider has been found in California, Nevada, Arizona, Utah, Colorado, New Mexico, Kansas, Oklahoma, Texas, Arkansas, Louisiana, Mississippi, Alabama, Georgia, Florida, Tennessee, North Carolina, South Carolina, Kentucky, Ohio, Virginia, West Virginia, Maryland, Delaware, Pennsylvania, New York, Massachusetts (Cape Cod) and New Hampshire, among other states. D'Amour et al.[4] reported that the black-widow spider is greatly increasing in numbers and invading the large cities. These spiders are found in greatest numbers in the vicinity of human habitations, not only in outdoor privies, as was formerly believed, but also in beds, garages, automobiles and tents and even high in office buildings.[5]

Although limited in practical application to regions where this spider is indigenous, knowledge of the clinical entity is important since many spider victims are subjected to needless operations because the symptoms often simulate acute surgical conditions of the abdomen. Almost every article written concerning the acute abdomen includes arachnidism to be considered in the differential diagnosis. If excruciating abdominal pain overshadows the rest of the syndrome surgical operation is often accomplished. An appalling record of human suffering has been checked back to *L. mactans* and its prototypes.[6]

The cases of arachnidism reported represent but a small fraction of the actual number that have occurred; many other cases noted by personal communications are never published. The arachnid menace is more extensive than one would expect. Its curtailment in an indigenous area can only be accomplished by unified efforts of mankind and parasitic enemy insects of the black-widow spider.

Evidence for the incidence of arachnidism rests largely on the statement of patients who have been bitten describing the insect answering the description of *L. mactans*. In other cases observers or the victim have caught the spider and brought it to proper authority to be identified.

In spite of the prevalence of the black-widow spider throughout North, Central and South America and especially in the southern half of the United States, relatively incomplete knowledge existed concerning its life history until the work of Blair.[7] The species *L. mactans* have been found in dark corners and in clothes closets. They are cannibalistic, feeding on each other whenever the opportunity presents itself. The nickname "black widow" given to the female of the species arises from its habit of capturing and feeding on the much smaller male after he has served the ends of species preservation.

The globose abdomen of the female stands out like a highly polished black pearl. It is attached by a slender pedicle to the smaller cephalothorax. The body averages 1.27 cm. in length. The abdomen is about 0.9 cm. in length. Slender pointed legs when expanded have a span of 3.8 to 5.1 cm. Legs and body are a glossy black and are covered by short, black hairs. On the ventral surface of the abdomen there is a rich red marking resembling an hourglass. Dorsal to the spinnerets in the midline of the convex

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surface of the abdomen is an additional red marking.

The full-grown female, particularly when distended with eggs, appears from experiments with animals to be the most poisonous. It is, however, a timid creature and when disturbed makes every effort to escape; this explains the relatively few bites in spite of the prevalence of this arachnid. When cornered or compressed, as between skin and clothing, the spider bites in self-defense. The male is ignored as an etiologic factor of any importance because of its size, timidity and scarcity.

The potent nature of the venom is readily appreciated when one sees a victim of the black-widow spider about an hour after a bite. The victim writhes in agony, terror-stricken, and expressing fears of death. Thesing[5] states that the venom of the female of this species is fifteen times as potent as that of a rattlesnake. The venom has been stated to be a toxalbumin with its most damaging activity on nerve endings. It is a thick, translucent, oily, lemon yellow-colored fluid, acid in reaction, from which a hemolysin and arachnolysin have been isolated.[8] A report by Macchiavello,[9] showing the results of spontaneous or induced stinging of guinea pigs by spiders, is indicative of the formidable nature of *L. mactans* (Table 1). The bite of the *L. mactans* produced the same nervous symptoms in the guinea pig as it did in man, death occurring between four and eighteen hours.

TABLE 1. *Effects of Spontaneous or Induced Stinging of Guinea Pigs by Spiders.**

| SPECIES | NO. OF EXPERIMENTS | NO. OF ANIMALS STUNG | NO. OF ANIMALS WITH SYMPTOMS | | NO. OF DEATHS |
|---|---|---|---|---|---|
| | | | LOCAL | GENERAL | |
| *Loxosceles laeta* .... | 75 | 28 | 28 | 0 | 0 |
| *Dysdera maxima* .. | 50 | 7 | 3 | 4 | 4 |
| *Metozygia dubia* .. | 20 | 0 | — | — | — |
| *Cleocnemis junior* .. | 23 | 0 | — | — | — |
| *"Migala"* ..... | 3 | 3 | 3 | 3 | 3 |
| *Zilla x-notata* ..... | 30 | 2 | 0 | 0 | 0 |
| Theraphosid ..... | 12 | 9 | 0 | 9 | 9 |
| *L. mactans* ..... | 25 | 18 | 3 | 18 | 14 |

*Adapted from Macchiavello.[9]

Other authors cited by Blair[3] have found similar results in experiments with warm-blooded animals. Experimental studies by Blair showed marked reactions in mice, rats, guinea pigs and chickens. In mice the mortality was practically 100 per cent. Rabbits, cats, dogs and sheep seemed little affected. Baerg[10] gave experimental evidence when, on July 9, 1922, after several attempts, he induced a black-widow spider to bite his finger for about five seconds, sharp pain in the finger and hand being the most prominent symptom, followed by aching pains in the muscles of the lumbar region, shoulders, chest and legs.

It was Blair,[3] in 1933, who allowed a black-widow spider to bite him in an experimental study and recorded careful clinical observations dividing the clinical picture into three stages: the stage of lymphatic absorption of the injected venom, characterized chiefly by pains in the bitten area and absence of systemic effects; the stage of vascular dissemination, characterized, clinically, by the explosive onset of widespread agonizing muscular pains and a condition of profound shock depending on the quantity of the venom or its toxic products; and the stage of elimination of the venom or its toxic products, characterized clinically by hypertension, diaphoresis, gradually diminishing muscular pain and evidence of renal damage — this stage is suggestive of an acute toxic nephritis.

## REVIEW OF CASES

The syndrome, as presented by 6 patients bitten by the black-widow spider observed in a seven-day period in an overseas tropical area, usually followed a similar pattern: transient excruciating local pain at the site of the spider bite; rapid local edema and redness of the skin at the site — in 2 cases the site could not be identified; in ten to fifteen minutes a "burning sensation" that spread centrifugally from the site of the bite and soon involved the whole body, passing off in about twenty to thirty minutes; a sudden abdominal pain, often cramp-like as in an acute surgical condition of the abdomen; cramp-like pains in the legs, arms and back; a general feeling of "utter weakness"; restlessness and extreme fear reaction, often hysteria; headache, nausea and vomiting; and burning of the soles of the feet (in unknown types of bites this symptom may be pathognomonic).

In children there may be convulsions, which are extremely difficult to control.[11] Other possible symptoms are variable such as paralysis, cyanosis, dyspnea and urinary retention.

Physical examination in this series revealed the following: the site of injection usually showed an area of erythema with mild edema (in 2 cases the site was not evident); a board-like abdomen, nontender to palpation, was present in all cases; there was hypersensitivity of the skin; the calf muscles were tender to palpation; 2 patients were in profound shock with blood pressure unobtainable (the other 4 patients had normal or slightly elevated blood pressures); motion of extremities was limited by muscle spasm, and flexion was a prominent feature; the temperature was normal or only slightly elevated; the pulse was slow, being 80 or under in all cases; examination of the blood showed a moderate leukocytosis; and the 2 patients presenting a picture of profound shock showed albuminuria.

The victims all gave an excellent history, and in all cases the spider was identified. One patient described his episode as follows:

> I was putting on my shirt this morning when I felt as if someone had stuck a needle in my shoulder. When I tore off my shirt a black spider with a red-orange spot on its belly fell out. The place stung for about 10 minutes,

and then a burning sensation spread all over my arm and soon over my whole body. I began to get stomach cramps and my legs ached. Soon I felt weak and dizzy and wanted to sleep but headache and a feeling of terrible nausea came over me.

The patients in this series were immediately given 10 cc. of 10 per cent calcium gluconate intravenously. Subsequently they were given a saline infusion containing 10 cc. of 10 per cent calcium gluconate. An ice bag was applied to the affected area. Relief was obtained in a short time in all cases and was followed by profound sleep. The patients were out of bed the next day and back to duty on the fourth day. Even the 2 patients in profound shock responded. No morphine was used, and antivenin was not available.

## Discussion

The venom has previously been reported to have injurious effects on the isolated heart of the frog.[12] Billman[8] reported a case with suggestive changes in the electrocardiographic tracing and recommended that more emphasis be placed on the cardiac status in arachnidism. Precordial and epigastric oppression are not uncommon. Bradycardia has frequently been noted by other observers.[2, 13, 14] In this series normal pulse rate or bradycardia was common.

Blair[3] believes that the condition of shock, characteristic of the second stage, suggests the presence of a histamine-like substance in the spider venom. Such a possibility is enhanced by the secretagogue action, evidence of contraction of the bronchial and intestinal musculature and symptoms of acute prostration noted in animal experiments. Indeed, Essex and Markowitz[15] observed certain histamine-like properties of another secretion, crotalin (rattlesnake venom), in experimental studies.

The clinical picture of the development of an acute toxic nephritis has caused much speculation. Other observers[11] considered the syndrome to be like hypertension in the adult and eclampsia in the child (children often have convulsions in arachnidism). Four of the 29 cases reported by Walsh and Morgan[13] and 2 of this series showed albuminuria. These findings may be similar to the "allergic nephritis" seen after bee stings.[16]

Another clinical syndrome noted in arachnid poisoning is cutaneous arachnidism, or the gangrenous spot of Chile.[9] Three types of arachnidism have been noted in Chile: nervous arachnidism, produced by the poison of *L. mactans*; cutaneous arachnidism or gangrenous spot, caused by the spider *Loxosceles laeta*, which injects a local necrotizing poison; and viscerocutaneous arachnidism, the intermediate type, in which the patients, in addition to cutaneous signs, showed symptoms of hepatitis with jaundice, hematuria, fever and slight nervous disorders. *L. laeta* is a house spider. There is often a painless latent period after the bite of the black-widow spider, but this never occurs with cutaneous arachnidism. There is absence of general symptoms in the "gangrenous spot of Chile," but the local necrotizing lesions are severe. The bite is followed by immediate changes in the surrounding epidermis, with blister formation, rupture and gangrenous area. The eschar sloughs, and a superficial ulcer forms and heals slowly.

## Treatment

About sixty preparations have been used in the treatment of arachnidism. Alcohol seems to be contraindicated, and fatal results have been reported from this agent.[11] Strychnine used to be given, and sedatives seemed the most rational treatment in former years. Intravenous magnesium sulfate until symptoms of spider poisoning have disappeared has long been recommended and seemed rational in cases in which hypertension was a prominent factor. Frawley and Ginsburg[11] had good results with magnesium sulfate in 11 cases, with freedom from symptoms in twenty-four hours. Hypertonic glucose has been used with varying results.[13] Morphine sulfate in heavy dosages has frequently been relied upon for relief by some physicians. The results of Gray's[17] observations with convalescent serum seem to bear out the suggestion that antivenin is developed by the patient recovering from the bite of the black-widow spider. It is reasonable to suppose that it will prevent symptoms or cure spider-bite poisoning. Antivenin (*L. mactans*) is listed in *New and Non-Official Remedies*. It is standardized on the basis of its ability to neutralize the venom of the black-widow spider when the two are injected simultaneously in mice. Bell and Boone[18] reported a single case in 1945 in which they regarded neostigmine methyl sulfate as an apparent specific treatment for arachnidism. This article stimulated much adverse comment from other observers, since much time and many other remedies had been given before neostigmine methyl sulfate therapy was instituted. It remained for Gilbert and Stewart[19] to propose calcium salts as a very effective method of therapy. They reviewed previous therapeutic measures in the treatment of arachnidism and presented 5 cases in which intravenous solution of 10 per cent calcium gluconate gave instant and prolonged relief of pain and also produced relaxation of muscular spasm. The intramuscular route was recommended in children, with almost immediate relief. Calcium lactate was ineffective orally, probably because of its incomplete and slow absorption. Calcium chloride is not recommended because of its possible necrotic action on tissues. In the series reported in this article intravenous calcium gluconate was found to give immediate and prolonged relief of muscle spasm and pain in all cases, and it is believed that this is the best available therapy in conjunction with other supportive measures. Others have remarked on the relief given by frequent hot baths.[3, 10] In Russia hot baths are often used in the treatment

of spider bite due to the "Karakurt," a closely allied species. Finlayson[20] believes that tetanus antitoxin should be used in all cases.


### Summary

Arachnidism, a definite clinical entity in medicine, is reviewed, and 6 cases successfully treated with intravenous injection of calcium gluconate are discussed.

Arachnidism should be considered in indigenous areas in the differential diagnosis of the acute surgical abdomen. It may simulate appendicitis, perforated viscus, peritonitis or any other acute surgical emergency.

The syndrome following the bite of *Latrodectus mactans* may give symptoms referable to many systems of the body. Nervous, cardiac, renal, pulmonary and cutaneous manifestations may be present.

Burning of the soles of the feet may be pathognomonic in arachnidism.

One should be familiar with the clinical picture so that proper diagnosis is made and proper therapy instituted with avoidance of unnecessary surgical operations.


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# PERFORATION OF INFARCTED INTERVENTRICULAR SEPTUM, WITH PARTIAL HEART BLOCK*


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BOSTON


THIS is the report of an acquired interventricular defect that developed at the site of an infarct high in the septum and was associated with partial heart block and a systolic murmur. In previous comprehensive surveys in 1934 by Sager[1] and in 1943 by Weber[2] a total of 35 cases of interventricular septal perforations due to infarction were collected. Since that time, 18 additional cases have been reported. Prior to the case reported below there were reports of only 2 cases of infarcted septal perforations with clinically detected conduction disturbances of the heart.

The purpose of this paper is to present a case of perforated infarct of the interventricular septum, to comment on the pathogenesis of the associated conduction disturbance and to review the relevant literature.

### Case Report

A 72-year-old woman was admitted to the hospital because of semiconsciousness of 4 hours' duration. Ten years previously she had suffered an attack of "acute" indigestion associated with substernal pain and dyspnea. Since then there had been no recurrence of these symptoms although she had noticed swelling of the ankles for the past few months. On the day before admission she suddenly felt weak and vomited, and was forced to return to bed. On the morning of admission she again vomited and returned to bed, and was found several minutes later in a semiconscious state. She was in obvious respiratory distress for the first time. There had been no chest pain.

Physical examination revealed a thin, dehydrated, semiconscious, orthopneic woman. The skin was cold, damp and cyanotic. Examination of the heart showed the rate to be 54 beats per minute, with frequent irregular beats and enlargement to the left and right. A loud systolic murmur was heard at the apex, but no thrill was felt. The liver was slightly enlarged.

The blood pressure was 76/40.

An electrocardiogram (Fig. 1) showed second degree heart block, an abnormal form of the ventricular complex (depressed ST segments in Lead 1, elevated ST segments in Leads 2 and 3 and late inversion of the T wave in Lead 3), elevated ST segments in Lead aVF, and depressed ST segments in Lead VI-5 with unusually high T waves in Lead V3,4. These findings were interpreted as evidence of acute posteroseptal myocardial infarction.

The patient failed to improve and died quietly 5 hours after admission.

On post-mortem examination the most striking findings were in the heart. The coronary arteries were markedly calcific. The left coronary artery was widely patent though markedly sclerotic. The right coronary artery at a point

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3 cm. from its origin was obstructed for a distance of 0.5 cm. by a dark-purple, moist mass. Microscopical examination revealed that this was not a thrombus but rather an acute exudative reaction within an intima that was the seat of

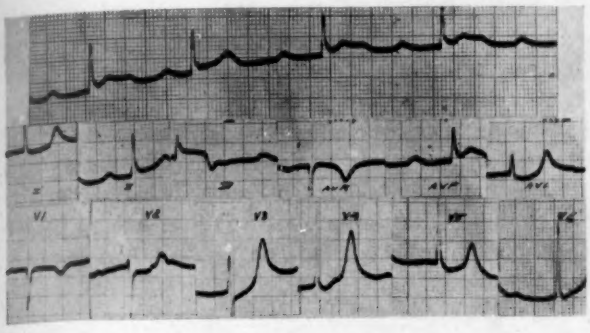

FIGURE 1. *Electrocardiogram, Showing Second-Degree Heart Block (Upper Strip) and Standard and Unipolar Extremity and Precordial Leads Demonstrating Changes of Recent Posteroseptal Myocardial Infarction.*

a severe and partially occlusive atheromatous degeneration (Fig. 2). The heart was sectioned transversely from the apex to the auriculoventricular sulcus at 1-cm. intervals. From the junction of the fibrous and muscular portions of the inter-

FIGURE 2. *Low-Power Photomicrograph of Coronary Artery, Which Grossly Appeared Occluded.*

*Note the marked exudate reaction and edema within the wall that is practically occluding the lumen.*

ventricular septum to a point 3 cm. distally, the myocardium was infarcted. Through the middle of this infarcted area there was a fistulous tract, 1 cm. in diameter, lined by fibrinous material connecting the two ventricles (Fig. 3). It is to be noted that except at the point of perforation of the infarcted septum there was a narrow zone of noninfarcted myocardium immediately beneath the endocardium and beneath the crest of the muscular portion of the septum. Microscopical examination showed changes corresponding to those of myocardial infarction of 48 to 72 hours' duration according to the criteria of Mallory and White.[3]

## DISCUSSION

In general, infarction involving the superior portion of the interventricular septum is likely to produce disturbances of conduction such as auriculoventricular block, whereas infarction of the lower apical portion of the septum is more apt to perforate, causing an acquired interventricular

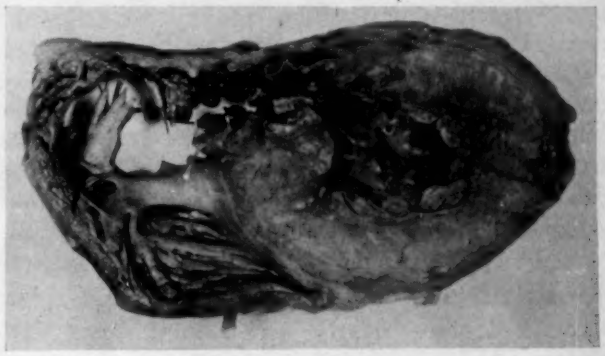

FIGURE 3. *Gross Cross-Section of the Heart near the Base, Showing Perforation through an Infarction of the Posterior Portion of the Interventricular Septum.*

septal defect and, characteristically, Roger's murmur. In the case reported above, the perforation of an infarct in the posterosuperior portion of the interventricular septum, with electrocardiographic evidence of second-degree heart block, comprised a situation of unusual rarity and interest. It was noted that the infarction extended to within 1 mm. of the crest of the muscular portion of the septum upon which, according to tradition, the auriculoventricular bundle of His rests. After coursing further forward, the bundle of His is said to divide into its two main branches, which then extend downward beneath the endocardium of the muscular septum to the apex. Thus, the location of the infarction and perforation in this case was such as to avoid the bundle branches, but may have involved the main bundle.

## REVIEW OF THE LITERATURE

The cases reported by Sager[1] and Weber,[2] as well as others, were confirmed by post-mortem examination.

In a series of 25,000 autopsies, Edmondson and Hoxie[4] found 72 cases of cardiac rupture (13 of which were septal) in 865 hearts with recent infarcts.

Wood and Livezey[5] described the case of a forty-four-year-old man who lived for four years and ten

months after septal perforation following myocardial infarction. This is the longest survival yet reported. It was the sixth case of a septal perforation following a posterior myocardial infarction and only the second case with the associated electrocardiographic finding of right-bundle-branch block.

A case from a clinicopathological conference was presented by Lober and Hertzog[6] in which an antemortem diagnosis of septal perforation was made.

The patient of Master and Russell[7] was a forty-seven-year-old man with an acute anterior and posterior myocardial infarction confirmed by the electrocardiogram who developed signs of septal perforation on the third day and died on the eleventh day. Autopsy confirmed these diagnoses and revealed a perforation in the inferior portion of the septum.

Lian's[8] case of posterosuperior perforation was diagnosed before death and was a remarkable combination of the typical clinical syndrome with electrocardiographic findings of acute posterior myocardial infarction. Analysis of expired air showed a low carbon dioxide content.

Diaz–Rivera and Miller[9] described 5 cases of cardiac rupture. Only 1 was septal, and it was recognized before death. The patient was a sixty-five-year-old man with typical infarction clinically. On the second day a systolic murmur developed at the cardiac apex and in the third interspace to the left of the sternum. The electrocardiogram showed right-axis deviation, multiple premature contractions and changes consistent with an acute myocardial infarction. Autopsy confirmed the diagnosis of septal perforation.

## Summary

A case of posteroseptal myocardial infarction in which rupture of the septum developed just beneath the "membranous septum" is reported. Electrocardiographic examination shortly before death showed second-degree heart block as well as the characteristic changes of posterior infarction. A loud systolic murmur was heard at the apex, but the patient was not observed long enough for the time of development of the murmur to be established.

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# PREMEDICATION IN ANESTHESIA

## Unusual Experience with Concurrent Administration of Barbiturate and Pantopon

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THE administration of an anesthetic may be smoothly supplemented or seriously distorted by the use of premedication. The importance of premedication in anesthesia manifests itself in two directions: it has a quieting sedative effect, which renders the patient more amenable to induction, reduces the amount of anesthesia needed for maintenance and prolongs the period of calm following operation; and it manifests side effects, particularly on respiratory activity, which may seriously disrupt the course of anesthesia. The action of the premedicaments in these respects is directly related to tissue susceptibility or idiosyncrasy on the part of the patient.

This report is a consequence of interest in the barbiturates as anesthetic agents that dates back to the use of sodium amytal over fifteen years ago, when a report on the use of that agent was published. In that report attention was called particularly to the undesirable side effect of the drug on the respiratory system — pulmonary edema. Four years ago major surgical procedures were undertaken with sodium pentothal administered intravenously as the principal anesthetic agent. A method of supplementing pentothal with transincisional novocain nerve block was worked out that established pentothal as a satisfactory anesthetic agent for intravenous injection. This statement is made despite the fact that the use of the agent was undertaken with a critical rather than a favorable bias after the unsatisfactory experience with sodium amytal.

The problem of premedication soon assumed basic importance. Pentothal is directly a depressant of the respiratory center, as are most other anesthetic agents. The depressing action is most marked on the expiratory side of respiration. Pentothal particularly suppresses the reflex stimulation of the expiratory phase of respiration, which results from neurogenic impulses originating in the field of operative trauma. In this respect it tends to restore the reflexly distorted inspiratory-expiratory time ratio from an abnormal 1:4 or 1:6 ratio to a more normal 1:1 ratio. However, pentothal does not exhibit a distinctive effect of both cyclopropane and ether in deeper planes of anesthesia — namely, paradoxical respiration. This effect depends on pharmacologic dissociation of the intercostal and diaphragmatic components of respiration, whereby deep inhalation anesthesia paralyzes the intercostal component long before there is paralysis of the diaphragmatic component. Pentothal anesthesia, in contrast, when carried too deeply produces concurrent intercostal and diaphragmatic paralysis with the sudden development of apnea. In the light of such pharmacologic facts, the use of premedicating drugs that initially depress the respiratory center calls for careful weighing of their value against the increased depression of the respiratory center by the anesthetic agent.

My experience with intravenous injection of pentothal and transincisional nerve block has persuaded me that cautious but effective premedication is a major factor in the successful and satisfactory use of pentothal anesthesia intravenously. The patients are calm, often very drowsy; neurogenic impulses, which reflexly stimulate the respiratory center and influence the rate of utilization of pentothal, are reduced in intensity; induction is more even and quick than it is in the absence of premedication; and the maintenance period requires lesser amounts of pentothal. In view of these apparent facts, I have been alert to the problem of synergistic or additive action of opium derivatives used in conjunction with intravenous administration of pentothal anesthesia. The following unusual case, therefore, was considered sufficiently illuminating on this point to merit reporting.

### CASE REPORT

A 61-year-old man was referred for surgical treatment of a partially obstructing duodenal ulcer, which had not responded to medical treatment. Operation had been planned under continuous penthothal and ether anesthesia with anterior splanchnic block. The patient was given the usual premedication therapy — 0.2 gm. (3 gr.) of nembutal by mouth the night before operation, 0.1 gm. (1½ gr.) of nembutal by mouth the morning of the operation, and 0.02 gm. (⅓ gr.) of pantopon with 0.0006 gm. (1/100 gr.) of atropine subcutaneously ½ hour before removal to the operating room. The operation was postponed after premedication had been given because of a reported temperature of 101°F. due to an upper respiratory infection. It was later noted that the patient became profoundly drowsy after the medication, with a respiratory rate of less than 10 per minute. After several days, when the cold was better, a posterior gastroenterostomy was performed under the following unusual circumstances. The premedication mentioned above was repeated. Intravenous administration of pentothal had just been started, and only 2 cc. of a 1 per cent solution (⅓ of a grain, a negligible quantity) administered, when the anesthetist noted that the respirations were only 5 per minute. The pentothal anesthesia was immediately stopped, and no further anesthesia of any kind was administered. The respiratory effort although infrequent was of full amplitude and vigor, and kept the patient in good color. The patient was in deep sleep. The incision was attempted and was continued through all layers of the abdominal wall until the peritoneum was opened. The patient slept soundly with no motor re-

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sponse or objective sign of discomfort from the incision. There was no reflex stimulation of the respiratory center from neurogenic impulses generated by surgical trauma, even from manipulation of the peritoneum such as traction on the stomach. The rectus muscles were relaxed by transincisional novocain block of the intercostal nerves in the sheath. This produced no sensory anesthesia. The anterior splanchnic block was not made, on purpose, the full extent of this unusual drug action being watched for. Intermittent administration of oxygen was carried out. The respirations continued at 5 per minute and were of full amplitude and force on the inspiratory side. Expiration was passive, with no reflex activation of the lateral abdominal muscles as accessory muscles of respiration. There was a long pause, resulting from total respiratory inactivity between expiration and inspiration lasting for about 10 seconds. As the patient's color and condition remained excellent the operation was continued, and a posterior gastroenterostomy performed in a perfectly quiet abdomen.

There was never the slightest evidence of discomfort on the part of the patient. He simply slumbered on. After completion of the anastomosis the abdomen was closed in a routine fashion without the slightest grimace or evidence of discomfort. While the skin was being sutured the patient was shaken sufficiently to arouse him slightly. He was asked whether he had any pain. He shook his head. From then on he made a slight grimace each time the needle was passed through the skin, but nothing else was observed. At the completion of the operation the patient was returned to bed and placed in an oxygen tent. His respirations gradually increased in frequency until they reached 12 per minute by 3 p.m. He became extremely restive at that time and was given 8 mg. (⅛ gr.) of morphine, which immediately depressed his respirations to 10 per minute. He made an easy recovery, being out of bed the following day. He returned home on the 14th day.

Additional observations made on the operating table and during convalescence enhance the interest in but fail to explain this unusual experience. During the course of the operation the patient was rebreathed with oxygen and carbon dioxide (95 and 5 per cent) with an increase in the volume but not in the rate of respiration. He was subsequently rebreathed with pure carbon dioxide for a few seconds, the volume of respiration again increasing and the rate rising from 5 up to 7 per minute. When the pure carbon dioxide was discontinued the rate of respiration immediately dropped back to 5 per minute. The patient was given irritating inhalations of ether fumes to determine if this stimulation would increase the rate of respiration, but it failed to do so. He was given an injection of metrazol and 0.4 mg. (1/150 gr.) of atropine without stimulating effect on the center; a repeated intravenous injection of metrazol and 0.4 mg. of atropine was given, but there was not the slightest increase in either the rate or the depth of respiration, even though the injections stimulated the cerebrum sufficiently to arouse the patient slightly.

During the period of convalescence an attempt to place responsibility for this effect was made by fractionation of the premedication. On one occasion 0.2 gm. of nembutal was given at night and 0.1 gm. repeated in the morning. The pantopon was withheld. There was no effect other than that of providing a good night's sleep. While the patient was resting in bed the respirations were 16 per minute, and he was quite awake. He was up and about shortly. On another occasion 0.02 gm. of pantopon was given in the morning with full precaution to avoid any administration of nembutal the night before. This drug alone had no apparent effect: the patient lay in bed fully awake, with respirations of 16 per minute. On a third occasion the entire premedication routine, with the exception of atropine and pantopon, was repeated with careful watch of the sequence of events. He lay quietly in bed, with respirations normal, awake and complaining because his breakfast was held up. He then received the hypodermic injection of 0.02 gm. of pantopon. Over a period of 10 minutes he gradually passed into a state of profound sleep from which he was aroused with difficulty. His respirations dropped steadily during this period to the depressed point of 9 inspirations per minute, and they remained at that level for about 1 hour. They then gradually returned to normal, and the patient was up and about in 4 hours, looking for his breakfast.

## Discussion

This experience was apparently a manifestation of unusual combined action by ordinary doses of nembutal and pantopon administered to an adult normal in all respects except for some loss of weight. On the basis of either synergism or additive effect an abnormal suppression of the usual controls of inspiration occurred. On separate occasions combined medication dropped respirations to 5 and 9 per minute. The failure of response to carbon dioxide places the effect on a neurogenic rather than a chemical basis. Failure of response to the pharmacologic stimulation of atropine and the analeptic metrazol indicated the profound depression of the nervous elements in the respiratory center. The absence of reflex neurogenic stimulation from the trauma of surgery was impressive. The phenomena were not in line with the usual action of barbiturates as noted in the administration of intravenous pentothal anesthesia. Barbiturate action tends to normalize the distorted respiration caused reflexly by neurogenic impulses originating in the operative zone. These pain impulses convert expiration from a passive to an active phase characterized by forceful expiratory contractions of the lateral abdominal muscles as accessory muscles of respiration. The expiratory phase of respiration is prolonged by these reflexes, with a distortion of the inspiratory-expiratory time ratio from the normal of 1:1 to an abnormal ratio of 1:4 or 1:6. Barbiturates (pentothal) act on the expiratory component, suppressing the sharp reflex activation of this expiratory phase until the ratio is restored to the quiet, normal 1:1 ratio of the resting state. Beyond this point, there is a reduction in the amplitude and force of respiration by the barbiturates, but usually no alteration of the rate even up to the point of complete apnea.

In contrast with the usual barbiturate action on the expiratory side this experience was one of ineffective inspiratory stimuli that failed to act on the respiratory center in a normal rhythmic fashion. There was a full normal inspiratory effort, followed by passive expiration. There followed a long pause for a resting period of nine or ten seconds. Predicating inspiratory act on the Hering–Breuer reflex, it seems that the threshold for these reflexes had been so elevated by the combined effect of the drugs that it required a summation of stimuli for a period of several seconds finally to achieve sufficient intensity to break over the elevated level of the threshold and create an effective inspiratory impulse. This is more in line with the action of the opiates, so that responsibility for this picture may be ascribed more to the pantopon than the barbiturates.

Undoubtedly, the reduced metabolism at sixty-one years of age contributed to this occurrence. As phenomenal as the depressing effect of these

drugs on the respiratory center was the interruption of the pain pathways to consciousness as a result of which a major operation on the upper abdomen was easily and comfortably performed without benefit of any other anesthetic agent. Administered separately, the drugs displayed no unusual effect. Administered concurrently, they exhibited a remarkable enhancement of pharmacologic effect in three directions: profound sleep, suggesting maximal barbiturate action on the cortex; interruption of pain sensations, suggesting pantopon and possibly some barbiturate action on the thalamus; and depression of respiration on the inspiratory side, suggesting enhanced pantopon action on the inspiratory neurons of the respiratory center — both intercostal and diaphragmatic.


### Summary

An unusual case of a gastroenterostomy performed under the effects of preanesthetic medication alone is reported.

Premedication consisted of 0.2 gm. of nembutal, given during the night before operation; 0.1 gm. of nembutal, administered on the morning of operation; 0.02 gm. of pantopon; and 0.0006 gm. of atropine, given half an hour before removal to the operating room.

Besides profound sleep and the absence of reflex response to pain, there was depression of respirations to 5 per minute. The respiratory center could not be stimulated by carbon dioxide in ordinary concentrations or by atropine, metrazol or irritating ether fumes; it responded feebly to pure carbon dioxide. It did not respond reflexly to the neurogenic stimuli of surgical traumatic origin.

Controlled postoperative observations showed that neither nembutal nor pantopon used separately in the same doses had any comparable action. In combination this profound effect, either synergistic or additive, was elicited on three separate occasions.

The possibility of such potent mutual enhancement of pharmacologic effect by barbiturate and opium derivatives administered concurrently prior to anesthesia should be fully recognized even though rarely encountered. This experience also emphasizes the care that should be exercised in the administration of narcotics postoperatively to patients who have received pentothal sodium intravenously during anesthesia.


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## CLINICAL NOTE

## AN OBSERVATION OF ANTITOXIN TITERS AFTER BOOSTER DOSES OF TETANUS TOXOID*


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THE efficacy of tetanus toxoid as an agent for the prevention of clinical tetanus has been well established.[1,2] The procedure now most widely employed is to give a basic immunizing course followed by a booster dose at least six months later. Another booster dose is then given in any emergency after an injury that might lead to tetanus infection.

The protection afforded is unquestionably based in large part upon the residual titer of antitoxin persisting after primary immunization and greatly augmented by routine booster doses. Much stress has also been laid on the capacity of an emergency dose, given after injury, to induce a rapid rise in serum and tissue antitoxin levels. Little information is available, however, regarding the rate at which this rise occurs in human beings. An opportunity arose of plotting the response to a booster dose in 5 members of the staff of the Division of Biologic Laboratories of the Massachusetts Department of Public Health, who received 0.5 cc. doses of fluid tetanus toxoid§ subcutaneously in the course of a routine immunization clinic in the Division. All five had previously been immunized with fluid tetanus toxoid either in the Division or while on military duty. The interval in each case since the last injection is shown in Table 1 below. Test bleedings were taken for determination of antitoxin titer just prior to this inoculation, and were repeated from the fourth to the tenth day, inclusive.

This study was undertaken as a result of the joint interest, extending over the past six years, of the Division of Biologic Laboratories and the Department of Bacteriology and Immunology of Harvard Medical School in the preparation, properties and use of tetanus toxoid, and is reported by those re-


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§The toxoid was prepared in February, 1942, from toxin produced in casein hydrolysate medium by Professor J. Howard Mueller at Harvard Medical School.

sponsible for the clinical and laboratory aspects respectively.

The serum antitoxin titers found are presented in Table 1 and Figure 1.

only 1 person in 8 whose titer started to rise on the fourth day. This present series follows more closely the immediate changes in titer, by means of daily bleedings.

TABLE 1. *Antitoxin Titers following Booster Doses of Tetanus Toxoid.*

| SUBJECT | INTERVAL SINCE LAST DOSE OF TOXOID | TITER BEFORE BOOSTER DOSE | TITERS AFTER BOOSTER DOSE | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | AT 4 DAYS | AT 5 DAYS | AT 6 DAYS | AT 7 DAYS | AT 8 DAYS | AT 9 DAYS | AT 10 DAYS |
| | | *units/cc.* | *units/cc.* | *units/cc.* | *units/cc.* | *units/cc.* | *units/cc.* | *units/cc.* | *units/cc.* |
| G. E. ..... | 1 yr., 8 mo. | 1.0 | 1.0 | >1.0<2.0 | 2.0 | >2.5<5.0 | >7.5<10.0 | >10.0<12.5 | 12.5 |
| J. A. Mc. .... | 2 yr., 10 mo. | >0.75<1.0 | >1.0<2.0 | >1.0<2.0 | 2.0 | >2.5<5.0 | >4.0<6.0 | >4.0<6.0 | >4.0<6.0 |
| J. M. N. ..... | 1 yr., 9 mo. | >0.75<1.0 | 1.0 | >1.0<2.0 | >4.0<6.0 | >7.5<10.0 | >10.0<12.5 | >10.0<12.5 | >10.0<12.5 |
| E. R. S. ..... | 5 yr., 5 mo. | >0.2<0.25 | No sample taken | >0.5<0.75 | >2.5<5.0 | >7.5<10.0 | >12.5<15 | >15<20 | >15<20 |
| H. J. B. ..... | 5 yr., 8 mo. | >0.025<0.05 | >0.025<0.05 | >0.1<0.25 | >0.5<1.0 | >2.0<4.0 | >2.5<5.0 | >5.0<7.5 | >5.0<7.5 |

The person with the highest residual titer had received toxoid the most recently, and conversely the lowest residual titer was coupled with the longest interval.

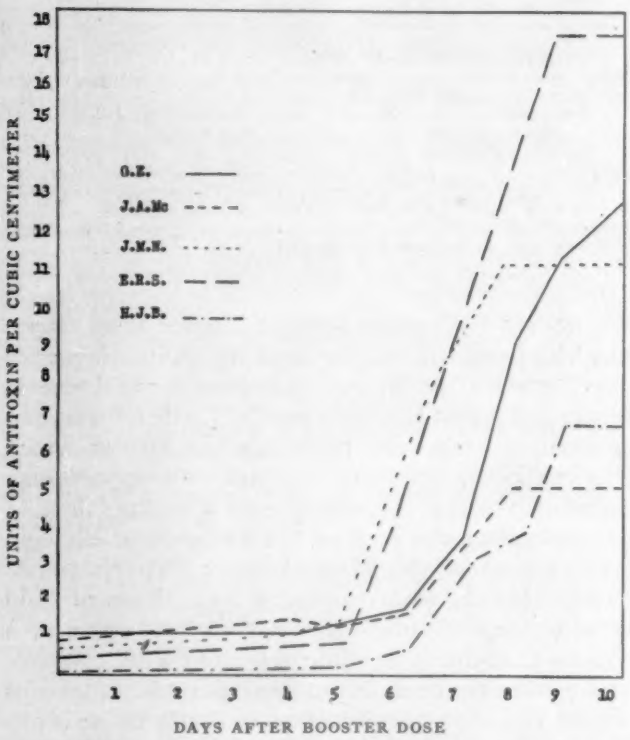


FIGURE 1. *Response to Booster Dose of Tetanus Toxoid.*

None of the titers started to rise before the fifth day, and all were leveled off or appreciably checked by the tenth day. This lag in rise compares with other findings such as those of Gold,[3] who found

Since many fatal cases of tetanus terminate within four days of the date of injury, it is apparent from this small number of data that dependence on the booster dose alone after injury may not assure sufficient protection. The residual antitoxin titer resulting from prior immunization may be of greater importance. For adequate protection against tetanus, therefore, it appears that patients should receive periodic maintenance doses as well as the primary immunization, and then receive emergency injections when injured. No set interval for such periodic doses has been established. Evidence suggests that definite protection may persist for as much as five years.[1, 4] In this study the residual titer of 1 subject (H. J. B.) at five years and eight months (Table 1) may have been below the level of adequate protection. Reason suggests, therefore, that periodic booster doses be given for maintenance purposes at intervals of three to four years.

The recent suggestion of a Massachusetts practitioner that physicians in the Commonwealth follow up former servicemen and servicewomen among their patients, and persuade them to have periodic tetanus booster doses, is an excellent one. If this is done a large group of the population can easily be kept immune to tetanus.

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# MEDICAL PROGRESS

## SURGICAL APPROACH TO CONGENITAL CARDIOVASCULAR DEFECTS*

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NEW ORLEANS, LOUISIANA

PROGRESS in the treatment of congenital cardiovascular defects has been so extensive and spectacular during the past decade that considerable interest has been focused on the entire field of cardiac surgery. In attempting a survey of the surgical approach to the special subdivision of congenital cardiac anomalies, it seems desirable to make a brief review of the older — as well as the new — aspects of general cardiac surgery to evaluate the recent advances more clearly.

### Historical Approach

The story of the surgery of the heart is one of the most fascinating in all the annals of medicine, although its development has been relatively slow. This was appreciated in the early days of this century, for Sherman[1] remarked in the annual surgical oration before the American Medical Association in 1902, "The road to the heart is only two or three centimeters in a direct line, but it has taken surgery nearly 2400 years to travel it." (The chest was first opened for empyema in the Hippocratic era.)

#### *Infection*

Acute suppurative disease was the first to receive attention,[2] although many years passed before the suggestions of the pathologists were acted upon by their surgical colleagues. Exactly three hundred years ago Jean Riolan,[3] though a staunch Galenist, and a violent opposer of Harvey's new teachings,[4] advised trephinement of the sternum for drainage of the pericardium. A full century passed; deSenac[5] described pericarditis, recommending drainage for its treatment with the opening in the chest to be made to the left of the sternum in an intercostal space. Still nothing was achieved, and after another half century Desault[6] and, a few years later, Larry[7] attempted the procedure, but unfortunately in both instances the diagnosis was in error. Romero,[8] in Spain, was finally successful in 1819, opening the pericardium for effusion in 3 cases, with 2 patients surviving.

Despite repetition in various countries, the operation did not gain favor, and Billroth[9] coldly stated, "Parencentesis pericardii is a prostitution of the surgical art . . . possessing more interest for anatomists than for surgeons." With this lack of encouragement it took surgeons nearly another century to pass from the pericardium to the epicardium across a space that is such only potentially.

#### *Trauma*

Louis Rehn,[10] of Frankfurt, was the first to suture successfully a wound of the heart, and he also was the first to perform decortication for adhesive pericarditis. Moreover, he accomplished a great deal of clinical investigation, and appeared frequently before the profession to defend the infant specialty. For these reasons he might well be considered the father of cardiac surgery. How young this branch of surgery really is can be appreciated by the fact that Rehn died in 1930[11] at the age of eighty-one. Interestingly enough, it was in his city of Frankfurt that William Harvey,[12] the discoverer of the circulation, first published (1628) his revolutionary studies on the motion of the heart and blood. To be sure, it was long before Rehn's time that Morgagni[13] had stressed the danger of compression of the heart from hemorrhage into the pericardial sac, which Rose[14] in 1884 termed "Hertztamponade." And Block,[15] of Copenhagen, confirmed Morgagni's observations that cardiac wounds were well tolerated and boldly proposed the suture of such wounds as a surgical remedy, having successfully demonstrated its feasibility on rabbits in 1882. Nevertheless, general response was not enthusiastic. Billroth,[16] in the following year — still cold — wrote, "The surgeon who should attempt to suture a wound of the heart would lose the respect of his colleagues." In 1896 Stephen Paget,[17] son of the famous Sir James Paget, stated, in the first work devoted to this subject in English: "Surgery of the heart has probably reached the limits set by Nature to all surgery: no new method and no new discovery can overcome the natural difficulties that attend a wound of the heart."

As if in rebuttal, Rehn,[10] that very year, reported the case of a twenty-two-year-old man with an actively bleeding wound of the right ventricle, which he closed with silk sutures; the patient made an uneventful recovery. This was all the spark that was needed.[18] Within six years, 34 additional operations had been reported. These were reviewed by Sherman[1] in an article that also described his investigations in the use of stay sutures in the ventricle as a method of holding the heart. In 1920 Sir

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Charles Ballance[19] gave an excellent historical review of the surgical treatment of infectious and traumatic aspects of cardiac disease.

*Acquired Disease*

Billroth[20] made another gloomy statement, around 1875, that "those most deeply concerned in the practice of internal medicine make plans for the boldest operations." If the surgical aspect of acquired cardiac disease is divided into three categories — valvular disease, adhesive pericardial disease and myocardial disease — operative intervention in each one was initiated by a bold, clear-thinking, prophetic clinician, each an internist who was to make significant contributions in other fields.

Sir Thomas Lauder Brunton,[21] the discoverer of amyl nitrite, startled the medical profession in 1902 with the suggestion that operative intervention be employed for mitral stenosis, but was uncertain whether the valve should be approached through the auricles or through the ventricles! Vigorous letters, pro and con, but mostly con,[22] appeared in the English medical journals. Nevertheless, American workers were intrigued by the idea, and pioneer investigative work was launched by W. G. MacCallum,[23] Harvey Cushing,[24] B. M. Bernheim[25] and the late Elliott C. Cutler.[26] Successful operations were reported by Tuffier[27] for aortic stenosis, and by Souttar[28] for mitral stenosis, both employing digital dilatation of the diseased valve. Cutler, Levine and Beck[29, 30] utilized a tenotome knife in 3 cases and then an ingenious instrument — a cardiovalvulotome[31] — to cut stenotic mitral valves in 4 more cases. Unfortunately, only the first patient survived. Allen and Graham[32, 33] perfected a cardioscope with which they could visualize the interior of the heart, but their clinical attempt to use it, as well as Pribram's[34] effort, met with failure. These experiences resulted in a quietus in surgical intervention for valvular disease until the present time.

Brauer,[35] who later established the modern concept of thoracoplasty, suggested the procedure of cardiolysis for adhesive pericarditis. His proposal gained favor much more quickly than that of Delorme,[36] who several years earlier, after careful study of cadavers, had advocated decortication of the heart. (He had previously introduced decortication of the lung.) Delorme pleaded in vain before various clinical societies in Paris for performance of his operation,[37] but it was not until Rehn, doubtful, but still a trail-blazer, performed a decortication for the severer internal form of adhesive disease, concretio pericardii, that the procedure was carried out. In 1920, 4 cases were reported with good initial results.[38]

Heberden[39] had described what he called "Pectoris Dolor," in 1772, and his contemporary, Jenner,[40] of vaccination fame, understood its underlying cause. Those so afflicted had to suffer almost another one hundred years before any effective therapy appeared, when Brunton,[41] just a year out of medical school, discovered that amyl nitrite could produce dramatic relief in anginal attacks. François-Franck,[42] known for his sign in patent ductus arteriosus and for cerebral localization, after twenty years of patient research, demonstrated that the sympathetic nervous system has a centripetal function, and that sympathetic ganglions are able to function as reflex centers.[43] He showed that aortic pain was carried by cervicothoracic sympathetic nerves and in 1899 suggested their resection in angina pectoris. An interval of seventeen years elapsed before Jonnesco[44] first put these ideas to a test. Daniélopolu[45] and Mandl[46] also attempted to relieve pain by resection or block of the sympathetic pathways.

The next attempt was to reduce the work of the heart by total thyroidectomy,[47] but this procedure has generally been abandoned.[48]

Beck[49–51] has been the most diligent worker in investigating means to improve the blood supply to the heart. He has tried, among other things: chemical irritation of the pericardium — Dakin's solution, silver nitrate, hypertonic saline solution and so forth; foreign-body irritation — gum acacia, horse serum, kaolin and bone powder; myocardial grafts — pericardial fat, pedicle grafts of skeletal muscle from the chest wall, the omentopexy of O'Shaughnessy[52]; and arteriovenous anastomosis (discussed below).

*Congenital Defects*

The chapter on congenital defects begins with J. C. Munro,[53] who proposed ligation of the patent ductus arteriosus at a meeting of the Philadelphia Academy of Surgery in 1907. This illustrious man was far ahead of his time, although Doyen,[54] in 1913, unsuccessfully attempted to overcome a congenital pulmonic stenosis with the use of a tenotome. In 1937 Strieder[55] was also unsuccessful in operating upon a patient with patent ductus arteriosus and superimposed subacute bacterial endarteritis.

Matas,[56] in his extensive treatise on cardiovascular surgery, did not even mention congenital defects, and only a decade ago, Levine[57] stated in his excellent text, "There is no known treatment for congenital heart disease." With timing reminiscent of Rehn, Robert Gross[58] introduced the present period with the first successful operation for congenital cardiovascular disease, ligating a patent ductus arteriosus in a seven-year-old girl in August, 1938. As might be suspected, one of Billroth's "internists," the child's pediatrician, J. P. Hubbard, had urged the performance of this procedure.

Other leaders of the present decade are Crafoord,[59] of Stockholm, who first operated for coarctation of the aorta in October, 1944, and Blalock,[60] who devised the procedure for congenital pulmonary

stenosis and first performed this operation in November, 1944. The list of cardiac firsts for this period is so full and varied that objective appraisal is difficult, to say the least.

Two women played a prominent role in laying the spadework for the surgical approach to congenital cardiovascular defects: one, a pediatrician, Helen Taussig, and the other a pathologist, Maude Abbott. Taussig made the suggestion to Blalock that an operation devised to increase the flow of blood to the lungs in cases of pulmonic stenosis would improve the condition of the so-called "blue baby."[61] Her recent monograph on congenital malformations of the heart is a classic.[62] After encouragement by William Osler, Maude Abbott[63] began a collective series of congenital heart lesions, and in 1936 published her atlas of 1000 cases, a milestone in cardiac history. Her efforts, more than anyone else's, provided the stimulus toward present-day interest in the treatment of cardiac anomalies. She devised a clinical classification for congenital heart disease in which patients are divided into three groups.[64]

Group I comprises those without abnormal communications between the right and the left sides of the heart — thus, cyanosis is not a part of the picture. Included are such congenital malformations as simple dextrocardia, pericardial anomalies, pure aortic or mitral stenosis and coarctation of the aorta.

Group II embraces patients with arteriovenous shunt, in whom arterial blood enters the pulmonary circulation. Cyanosis is usually not observed. There is, however, possible transient reversal of flow, with cyanosis. Contained in this group are defects of the interauricular or interventricular septums and patent ductus arteriosus.

Group III includes patients in whom cyanosis is a prominent feature. Among the many causes are defects of the interventricular septum with dextroposition of the aorta, tricuspid stenosis and atresia, transposition of the great vessels, Eisenmenger's complex and the tetralogy of Fallot.

It is exceedingly difficult to estimate the incidence of these major groups or of their components, because of the multiplicity of many defects, as well as the variations that occur with a single lesion, for example, an interauricular defect may be high, low, large, small or primary, or may represent persistence of the foramen ovale and so forth. Nevertheless, it is desirable to have some idea of the approximate range of occurrence, as an aid in orientation, if for no other reason. Since no estimate was noted in the literature surveyed, a list of major defects, admittedly not exact, was compiled on the basis of an analysis of Abbott's cases (Table 1).

Table 1. *Compilation of Major Defects.*

| Defect | Incidence % |
|---|---|
| Acyanotic group (approximately 25 per cent) | |
| Coarctation of aorta | 8 |
| Anomalies of aortic arch | 2 |
| Aortic stenosis | 2 |
| Anomalies of semilunar cusps | 5 |
| Pericardial defects | 2 |
| Displacements of heart | 2 |
| Cyanose tardive group (approximately 50 per cent) | |
| Patent ductus arteriosus | 13 |
| Defects of auricular septum | 20 |
| Defects of ventricular septum | 17 |
| Cyanotic group (approximately 25 per cent) | |
| Tetralogy of Fallot | 12 |
| Tricuspid atresia | 1 |
| Pure pulmonary stenosis | 1 |
| Transposition of great vessels | 2 |
| Eisenmenger's complex | 5 |
| Persistent truncus arteriosus | 1 |
| Complete septal defects (cor triloculare and biloculare) | 1 |

## Embryologic Approach*

Before the conditions amenable to surgery are considered, it is of interest to note the major embryologic implications of congenital cardiac defects. Developmental defects of the heart and great vessels are surprisingly few, considering that the mammalian embryo, having practically no yolk available as food, is dependent upon prompt establishment of relations with the maternal circulation. This entails the necessity of a precocious development of the vascular system of the embryo, for the maternal circulation remains confined within the uterine wall and the embryonic circulation must grow to it.[65]

The heart of the human embryo begins to be formed during the third week of development. During the third and fourth weeks the primitive cardiac tube can be seen passing through the pericardial cavity and fixed to the pericardial wall only at its caudal venous entrance and cephalad arterial exit. Since it grows faster than the pericardial cavity, it is bent first in a loop and then in a fallen-S-shaped curve to stay within its boundaries. The proximal arterial end bulges forward and downward, whereas the atrial end pushes behind and above the ventricle. The blood leaves this primitive heart by way of the truncus arteriosus. Later, the truncus separates into aortic and pulmonary trunks. The basis of the partitioning of the heart into right and left sides is largely laid down during the second month of development.

The critical period in the human subject occurs between the fifth and the eighth week — that is, before the cardiac septums are fully formed and while the complex processes of torsion, involution, readjustment and fusion are taking place at the base of the heart, interruption of which is the source of most of the graver anomalies.

If development stops with the pre-septate tubular organ with ventricular bend to the right, the adult fish heart is seen. Similar conditions prevail in the amphibian heart, except that here subdivision into lateral chambers is suggested. Both are examples of cor biloculare.

*Patten's excellent new text *Human Embryology* was used as a guide for this section.

Further ontogenesis is reflected in the reptilian heart, the ordinary snapping turtle having two completely divided auricles, but only a common ventricle — cor triloculare.

Meckel,[66] the "younger" of that famous family, noted in 1802 the curious manner in which graver cardiac anomalies mirror the hearts of the lower vertebrate orders. Von Rokitansky[67] (1875) placed the origin of septal defects in the early weeks of embryonic life, while in 1905 Sir Arthur Keith[68] showed that persistence of the reptilian bulbus was the main etiologic factor in pulmonary stenosis. Spitzer,[69] in 1923, crystallized these investigations with his theory that arrest or delay of the clockwise torsion that normally takes place in the tubular heart between the fixed arterial outlet and venous inlet during the process of septation accounts for transposition of the great vessels, inverted ventricles and most of the other anomalies.

## Etiologic Approach

The cause of malformations of the heart is not clear. Undoubtedly many factors may affect the development of the embryo, both intrinsic and extrinsic.

### *Intrinsic Factors*

These factors are inherent in the male or female germ plasm prior to fertilization, owing to defects in genes transmitted by the laws of heredity. These defects occur because of the phenomenon of mutation — that is, the occurrence of a change in one of the genes that compose the chromosome thread, becoming reproduced in subsequent generations.

The production of such anomalies is in a sense the most characteristic thing about all living matter, being the property that most basically distinguishes living matter from nonliving and that has allowed protoplasm to develop, in its evolution, all the further peculiarities of the human organism.[70]

Murphy[71] has estimated the occurrence of live-born, malformed offspring to be 1 in 213 births. There is a better than 10 per cent chance that a subsequent pregnancy will eventuate in a malformed infant (1 in 8, or twenty-four times greater than the average incidence). The great influence of intrinsic factors is represented by the fact that Murphy found in families possessing two or more malformed children, the defect in the first-born malformed member duplicated in a subsequent malformed sibling in nearly half the cases. This incidence of 50 per cent also was true of duplication of malformation in distant relatives.

The actual incidence of congenital cardiovascular defects at New Orleans Charity Hospital between 1927-1936 was 1.08 per cent.[72]

### *Extrinsic Factors*

Therapeutic amounts of radium or roentgen irradiation of the human pelvis during pregnancy are extremely likely to injure the embryo.[73] This injury may be due to breakage or rearrangement of chromosome parts, so-called "translocation," which has been shown to occur when mature germ cells are subjected to radiation. Muller,[74, 75] recent Nobel prize winner, has estimated that the extrinsic factor — irradiation — produces gene mutation (thus becoming an inherited or intrinsic factor) about one hundred times greater than the frequency of spontaneous mutation. It has also been established that the frequency of induced mutation will be proportional to the total dose of radiation received over an unlimited period — that is, the total amount absorbed. Because most such mutations are recessive many generations may pass before the eventual lethality of the defect appears or "shows." On this basis Muller has calculated, for example, that each atomic bomb falling in a highly populous area would probably kill more people scattered throughout future generations than those killed in the generation immediately affected.

Other mutagenic agents are mustard gas,[76, 77] carcinogenic agents[78, 79] and perhaps even cosmic radiation.[80] The importance of mutational prophylaxis as related to congenital defects is obvious.[70]

Virus infection has likewise been implicated as a major extrinsic factor.[81] Gregg[82] and Swan[83] have definitely shown that German measles in the first two months of pregnancy is associated with an extremely high incidence of congenital cataracts and congenital malformations of the heart; if maternal rubella occurs in the third month of pregnancy there is still a high probability that such defects will occur. After the third month the cardiovascular system is practically completely formed, and there is apparently slight liability of injury to the fetus.

Vitamin deficiency during pregnancy has also been shown to be a factor.[84]

## Diagnostic Approach

Taussig[62] has emphasized the differences in diagnosis of congenital heart disease of infants as compared to adults. The same methods of diagnosis that have proved useful in adult cardiac abnormalities were also applied to infants. This is a serious error. Whereas special characteristics of murmurs are of great diagnostic aid in adults, in infancy, although the appearance of a murmur may suggest an abnormality, the heart is too small, the blood pressure too low, and the chest wall too thin for the murmur to be of specific diagnostic aid. The existence of a congenital malformation of the heart is suggested in infants by the detection of unusual murmurs and thrills, by the presence of cyanosis with or without clubbing of the fingers and by alteration in the size or shape or position of the heart.

Accurate diagnosis is frequently made possible by the information derived from physical and x-ray examination, and fluoroscopy. Additional important

aids to diagnosis are angiocardiography — that is, contrast visualization of the heart and great vessels with opaque substances — and physiologic studies of intracardiac pressures, oxygen saturations and calculations of cardiac blood flow. Such measurements are obtained by catheterization of the right side of the heart, including the right auricle and the right ventricle and, when possible, the pulmonary artery. The chief criteria for diagnosis are alterations in oxygen content in one or another of these chambers and alterations in pressures.

Cournand and his associates[85, 86] have shown that by means of the Fick principle[87] the volume of flow through these chambers may be estimated. The output is calculated from the difference between the oxygen content of the venous blood and that of the arterial blood, and the total oxygen consumption. It is evident that if the quantity of oxygen that a unit of blood delivers to the tissues (or takes up from the lungs) is known, together with the total quantity of oxygen consumed over a given period, the volume of blood that had been engaged in the carriage of this quantity can be calculated.

For example, by an actual measurement of oxygen in femoral-artery blood a figure of 20 vol. per cent may be obtained. This means that 20 cc. of oxygen is present in each 100 cc. of arterial blood. After the catheter is known to be in the right auricle by pressure measurement and fluoroscopic check, a blood sample taken from the auricle (mixed venous blood) might show a figure of 15 vol. per cent — that is, 15 cc. of oxygen per 100 cc. of venous blood. In this hypothetical normal subject, the total oxgyen used by all the tissues in a minute's time might measure 300 cc. Thus, it is seen that 5 cc. of oxygen is taken up by the tissues per 100 cc. of blood while at the same time the subject is consuming 300 cc. of oxygen per minute; then 300 divided by 5 will give the units of blood in 100 cc. flowing from the aorta (the oxygen content being practically the same as that of the femoral artery) to the right auricle, or the total systemic blood flow. In this case it would be 60 units of 100 cc., or 6 liters per minute.

In the lungs the same principle holds, with the oxygen, of course, going into the blood, instead of going out. If the catheter tip is placed in the pulmonary artery a blood sample may be obtained that might show a figure of 15 vol. per cent, and that from the femoral artery (which is practically the same as the amount in the pulmonary vein) might again be 20 vol. per cent. This would show that 5 cc. of oxygen has been added to each 100 cc. of blood flowing through the lungs. Then the pulmonary flow in such a case is:

$$\frac{300}{20-15} \times 0.1 = 6 \text{ liters per minute.}$$

The volume of flow through single, one-direction shunts may most easily be estimated by calculation of the difference between the pulmonary and peripheral flows. If the shunt is in both directions more complicated formulas must be used.[88]

By means of such measurements, the anatomic lesions in most cases can be predicted with a great deal of accuracy. Nevertheless, because of the inherent difficulties in making an exact diagnosis and the necessity for the therapeutic team to know as much about each patient's status as is humanly possible, radiographic studies should also be utilized.

Though the technic had previously been described, Robb and Steinberg[89] placed angiocardiography on a practical basis. The usual procedure is to inject 50 cc. of double-strength diodrast as rapidly as possible in an antecubital vein. Multiple exposures are then taken, either manually or by use of special equipment, with the patient in an oblique position so that views will be obtained showing the bolus of opaque material in its passage through the various chambers of the heart, the lesser circuit, the great vessels and any abnormal communications that may be present (Fig. 1).

As suggested by Getzoff[90] we have been having our subjects ingest several tablets of an antihistamine drug a short while before the dye is injected, to minimize allergic reactions. The procedure has been found to have almost no ill effect and has proved extremely valuable in differentiating obscure lesions.[91, 92]

## Patent Ductus Arteriosus

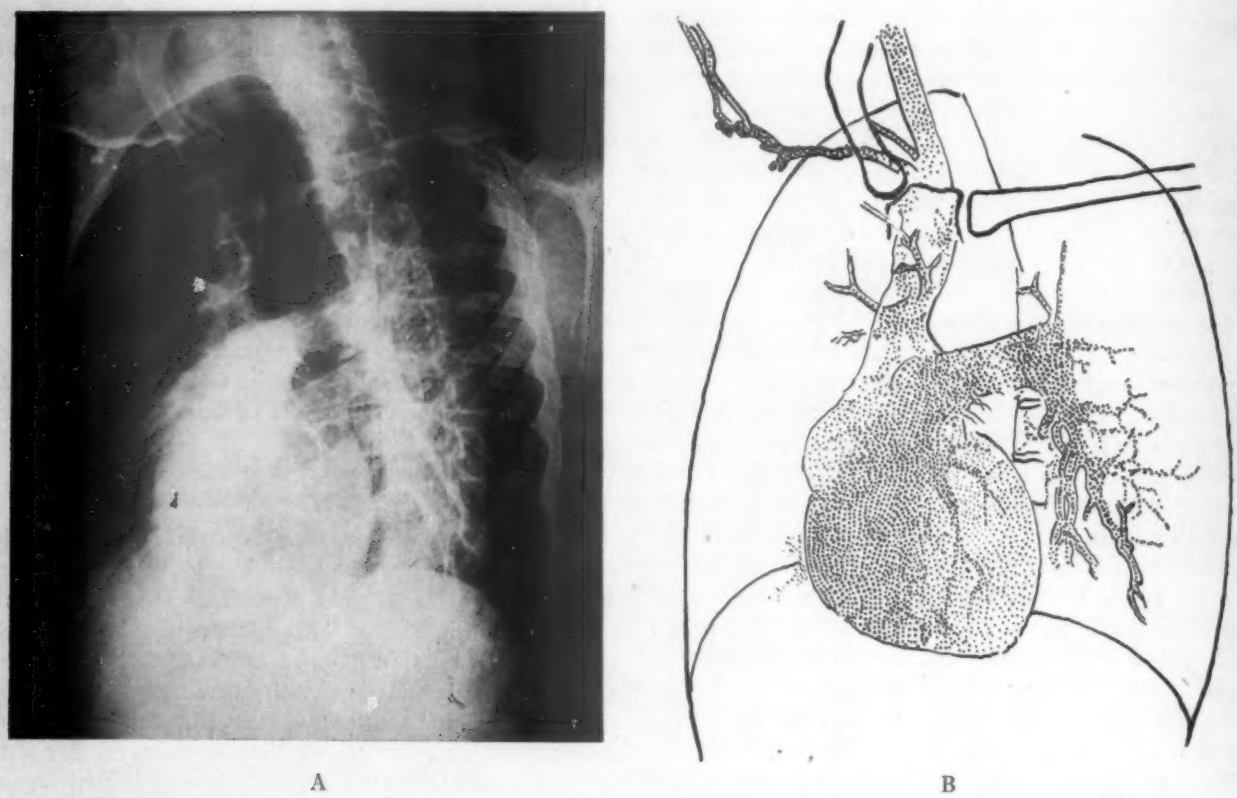
Patent ductus arteriosus, although originally noted by Galen,[93] has been known as the ductus Botalli since its alleged description by Botallus[94] (he was born in 1530). This curious by-play in medical history transpired when a charitable editor inserted an extra anatomic plate in a new system containing Botallus's observations, but long after his death.[95,96] Cristie[97] found that whereas 65 per cent of these vessels are still open at the end of two weeks of extrauterine life, only 1 per cent are patent after one year. It is estimated that there are approximately 20,000 people in this country with a patent ductus arteriosus. In Abbott's[98] review of 1000 cases, the incidence of patent ductus arteriosus was second only to that of intracardiac septal defects. In this malformation, as in a number of others, there is a curious sex distribution. For some unknown reason the defect is two or three times more common in females than in males.

Munro[53] demonstrated the feasibility of operation by performing ligations on newborn cadavers, but he stated that he tried in vain to inspire the pediatrician with his views. There still remains a difference of opinion regarding which cases should be selected for surgery. Taussig[62] states that in general the prognosis varies with the size of the heart and that in the vast majority of cases the heart is but slightly enlarged. She claims that the out-

look is good, the malformation being compatible with a long and active life.

At variance are the statistical data of Abbott,[98] Bullock et al.[99] and Keys and Shapiro.[100] Abbott's mean age of death in 92 cases was twenty-four years (including, however, 20 patients who died in infancy). Bullock and his associates reviewed the cases of 80 patients, all over the age of three years proved by autopsy to have no other significant cardiac anomalies. Half the patients died by the age of thirty, and 71 per cent were dead by the age of 40 years; 86 per cent died as a result of the complications intervenes. Very few survive once they develop failure (in marked contradistinction to patients with mitral disease).

Von Rokitansky[67] showed in 1852 that the direction of flow was from the aorta to the pulmonary arteries. This has been confirmed by Eppinger, Burwell and Gross[101] who have also demonstrated that the volume of the leak in patients with large shunts constitutes up to 75 per cent of the blood expelled by the left ventricle, and because of this large volume of blood returned to the left ventricle without passing through the right, the output of the



A B

FIGURE 1. *Coarctation of the Aorta — Reproduction and Tracing of Unretouched Angiocardiogram Taken Eight Seconds after Injection of Dye into the Antecubital Vein.*

*The right side of the heart and pulmonary arterial system are visualized.*

genital lesion — approximately half from bacterial endarteritis, and a fourth from congestive failure. Excluding cases in infants and those with cyanosis, the probability that a patent ductus arteriosus will be associated with other significant anomalies is relatively small.

Keys and Shapiro[100] analyzed 60 cases in adults (defined as persons over the age of seventeen) and found the life expectancy about half that of the population as a whole — roughly twenty-five years' reduction, or an average age at death of thirty-five years. They claim that in the majority of cases the patient's life is fairly normal while it lasts. Almost none are cardiac cripples. As a rule, they maintain good compensation until one of the com-

left ventricle may be two to four times that of the right.

Stunting of growth and underdevelopment are the results of the bypass of a large proportion of the oxygenated output of the left ventricle back into the pulmonary circuit. There is a compensatory increase in the total volume of circulating blood, both plasma volume and red-cell mass,[102] but this is usually inadequate to meet nutritive requirements, although it must be remembered that many persons with a small shunt never have any disability as a result of the abnormality. The diastolic pressure is usually low and upon exercise may drop to zero.[103] The pulse pressure is usually wide, depending upon the volume of the shunt. These findings

explain why with all but small shunts severe complications are prone to develop. Consequently, it is concluded that an attempt at ligation is justified in practically all cases, regardless of the absence of signs or symptoms of decreased adjustment to the defect.

The optimal time for operation is in childhood since adults are more likely to have less cardiac reserve, and frequently have an extremely short ductus arteriosus, which makes the operation more hazardous.[104] Gross[104] first did a ligation in continuity, and he stated that in children, when the ductus is soft, yielding, elastic and easily compressible, a ligation can almost always be relied upon to produce a permanent closure. In older patients, particularly in those beyond eighteen years of age, the vessel is frequently difficult to compress, and has an exaggerated pulsation transmitted to it by a sclerotic and fixed aorta. In these cases division and ligation, as first reported by Touroff,[105] have given best results.[106] Blalock,[107] who believes that routine division of the ductus arteriosus is an unnecessarily dangerous procedure, employs a method consisting of purse-string sutures at either end of the vessel, a through-and-through mattress suture between and a ligature of umbilical tape over the mattress suture.

Before the duct is tied, it should be compressed manually to be certain there is not an associated hypoplasia or stenosis of either great artery, in which case the ductus arteriosus functions as a necessary physiologic shunt.

Crafoord[108] reports that about half his 71 cases were ligated in continuity and about half divided. He has had only one recurrence (in a case in which the vessel was divided) and now covers the cut ends with a pericardial flap. There have been 2 deaths in his series.

Jones[109] prefers ligation in continuity and has operated in 61 cases with 2 deaths. He has also abandoned the anterior approach, used by most surgeons, and now employs a posterolateral one. An analysis of complications in his series revealed recurrence of murmur (10 cases), atelectasis (5 cases), empyema (2 cases), hemoptysis (2 cases) and paralysis of the recurrent laryngeal nerve (2 cases). Gross's[106] series numbered 180 cases at latest report, the ages varying from two to forty-six years, with 4 deaths. The average mortality in a series of 441 collected cases was 4.5 per cent.[106-113]

## Coarctation of the Aorta

Coarctation of the aorta was described by Morgagni[13] in 1760. Subsequently Bonnet[114] divided this anomaly into two groups: infantile and adult. The former is found in infants, most of whom die during the first year of life — the atresia generally involving the entire fetal isthmus of the aorta (the region from the left subclavian artery to the junction of the ductus arteriosus) and usually associated with other major cardiac anomalies. Fortunately, this type is relatively uncommon (less than 5 per cent) although a narrow descending arch is a normal occurrence in fetal development.

The adult type is a more focal area of constriction, usually 1 cm. or less in length, located most frequently at or just below the insertion of the ductus arteriosus. This type has been described in infants,[115] and the infantile type in adults,[116] but since the adult form has no embryologic counterpart in normal intrauterine life, it is quite likely that the two types are fundamentally different anomalies.

Abbott[117] searched all the available literature from the time of the earliest case report (1791) by Paris[118] to 1928, collecting 200 cases. Reifenstein, Levine and Gross[119] had collected 104 additional cases up to 1947. They found that this defect is only occasionally compatible with long life, at least 61 per cent of patients dying before the age of forty (in Abbott's series the figure was 74 per cent). The average age at death was thirty-five years (thirty-three and a half years in Abbott's series). Again, there is a curious sex predilection, the lesion being more common in males, 5:1 (in Abbott's series, 4:1). Improvement in ante-mortem diagnosis had occurred since Abbott's report — 40 per cent as compared with 14 per cent.

There is no evident relation between the degree of coarctation and the age at death or cause of death, which may be due to heart failure (18 to 29 per cent of cases), rupture of the aorta (20 to 23 per cent), bacterial endarteritis (16 to 22 per cent) or cerebral hemorrhage (10 to 12 per cent). Approximately a fourth of these patients die of incidental causes.

There does seem to be a direct relation between the degree of stenosis and the extent of collateral circulation, which produces the signs and symptoms of the disease and eventually the diagnosis. The majority of cases possess a grave degree of stenosis. A fourth of Abbott's patients were completely atretic, and in over 50 per cent of the remainder, the lumen was less than 6 mm. in diameter.

The cardinal diagnostic features[120, 121] include hypertension in the vessels of the upper extremity (frequently not marked); decreased pulse volume and blood pressure in the lower extremities, often associated with numbness and coldness; and collateral arterial anastomoses. These develop principally by way of the shoulder girdle (subscapular branch of the axillary artery, transverse scapular and cervical branches of the subclavian artery) and the anterior chest wall (internal mammary artery). As a result there may be pulsations or murmurs in unusual places, frequently interscapular or anteriorly adjacent to the sternum, or rib erosion[122] — this important finding generally takes several years to appear so that x-ray evidence of it is frequently lacking in children (Fig. 2).[123]

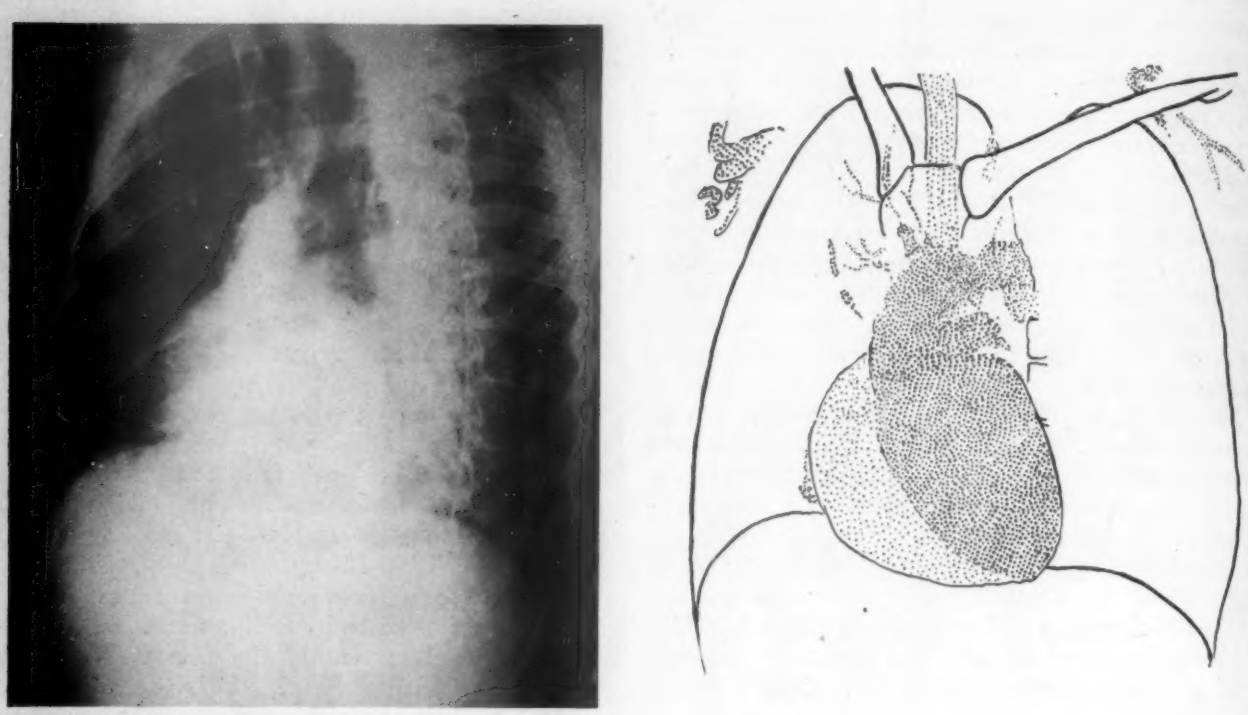
It should be emphasized that in the early decades of life, symptoms are usually absent, and a prompt

diagnosis will not be made unless there is a careful study of all young patients. Even then the clinical picture may be obscure, and in these cases contrast visualization of the great vessels is an important adjunct in the study of the patient.[124]

The first suggestion of definitive therapy was made by Blalock and Park.[125] They investigated the feasibility of anastomosing the subclavian artery to the aorta, distal to the constriction, and performed the operation on dogs, suggesting that the left common carotid artery might be a better vessel to use in human beings. Crafoord,[126] of Stockholm, reasoned that since the human subject had developed a tolerance to poor aortic blood flow distal to the stenosis, the aorta in these patients could be safely occluded for as long a period as was necessary to excise the stenotic area and do an end-to-end anastomosis. He performed the first two operations, both on male patients, in October, 1944 — one twelve years old and the other twenty-seven years old. Both survived.

Gross[127] independently had arrived at similar conclusions, and he was next to perform the operation in June, 1945. He uses cyclopropane anesthesia and has adopted a posterior thoracic approach through the bed of the fourth rib. Segments of the posterior ends of the third, fifth and sixth ribs are also removed in a vertical incision that connected the lateral one, forming a T. The vascularity of the region is tremendously increased, so that blood loss may be great. The aorta is dissected free and the ligamentum arteriosum cut, care being taken to avoid the left vagus and recurrent laryngeal nerve as well as the thoracic duct. After resection of the coarctation, the aorta is anastomosed with either one or two rows of sutures. Either interrupted sutures or a continuous U-suture may be used, the ends being everted so that intima is in contact with intima. The proximal clamp on the aorta must be released gradually over a period of five to six minutes to prevent circulatory failure,[128] since venous return to the heart may be temporarily diminished during the time that the depleted vascular system of the lower portion of the body adjusts itself to its increased blood volume. (Crafoord[126] states that he has had no difficulty with rapid release of this clamp.)

Clagett[129] has had to use the procedure originally suggested by Blalock because of technical difficulties, and in a discussion at a recent meeting it was stated that he has employed this procedure on five occasions with 1 death; the ages varied from eighteen to thirty-four years.[130] In each case severe degrees of aortic sclerosis were noted — a finding important to the thesis that all patients with coarcta-



A B

FIGURE 2. *Same Patient as That in Figure 1.*
*The time is five and a half seconds later. The dye has returned from the lungs and is visible in the left side of the heart, ascending aorta and great vessels of the neck. Note visualization of the coarctation of the aorta and dilated and tortuous subscapular vessels.*

tion should be operated upon early to obviate degenerative sequelae.

Operative intervention is the only definitive treatment, although the not infrequent concomitant finding of congenital intracranial aneurysm (5 to 10 per cent of cases) and bicuspid aortic valves (25 to 40 per cent) militate somewhat against the hopeful results to be expected from surgery. The optimal time for operation is in the second decade.

Below the age of six or seven the aorta is generally too small to work on satisfactorily, and over the age of twenty-five to thirty, it is apt to be sclerotic above the obstruction or thinned out below. Gross[131] reports 40 explorations, of which 35 were resectable. There were 5 deaths. There has been a diminution of hypertension in the upper extremities to normal in all but one case associated with increase in pulsation and rise in arterial pressure of the leg vessels.

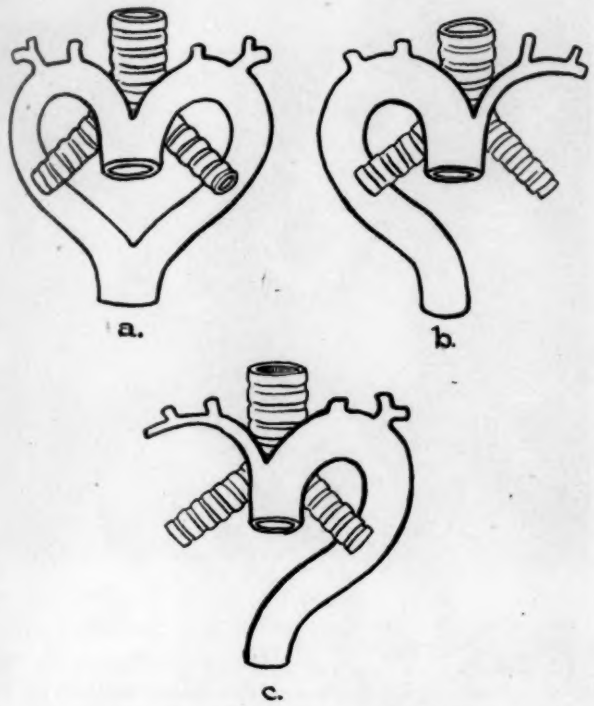



FIGURE 3. *Comparative Anatomy of the Aortic Arch.*

*A = retention of primitive double aortic arch — found in frogs, cause of so-called "aortic ring" in human beings. B = retention of right aortic arch, a normal occurrence in birds (seen in approximately 20 per cent of cases of tetralogy of Fallot). C = arching of aorta to the left, the usual occurrence in the human being (many variations of these three basic types of development may be encountered).*

## PULMONARY STENOSIS AND ATRESIA

Stenosis or atresia of the pulmonary artery was present in 150 of 1000 cases reported by Abbott.[98] About 25 per cent were not associated in the manner described below. The remainder were generally classified as cases of tetralogy of Fallot — that is, pulmonary stenosis or atresia, defect of the interventricular septum, dextroposition of the aorta (discussed below) and hypertrophy of the right ventricle. This is the most frequently encountered type of congenital cardiovascular defect associated with cyanosis.

Operative experience has led Blalock[132] to emphasize the fact that in approximately 20 per cent of operative cases the aorta will be seen arching to the right instead of its usual course to the left, and descending on the right side rather than the left (Fig. 3). Thus the aortic arch and descending aorta are dextroposed. This has given rise to an ambiguity in terminology, and, as suggested by Rives,[133] it is urged that the orifice of the aorta be referred to as dextroposed when the tetralogy of Fallot is described. As a matter of fact, the fault

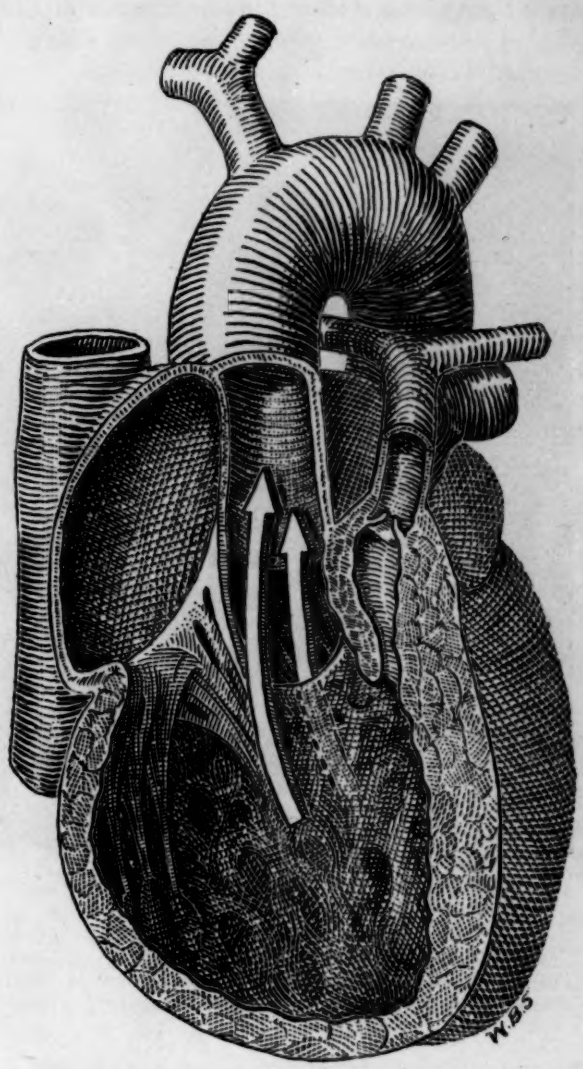
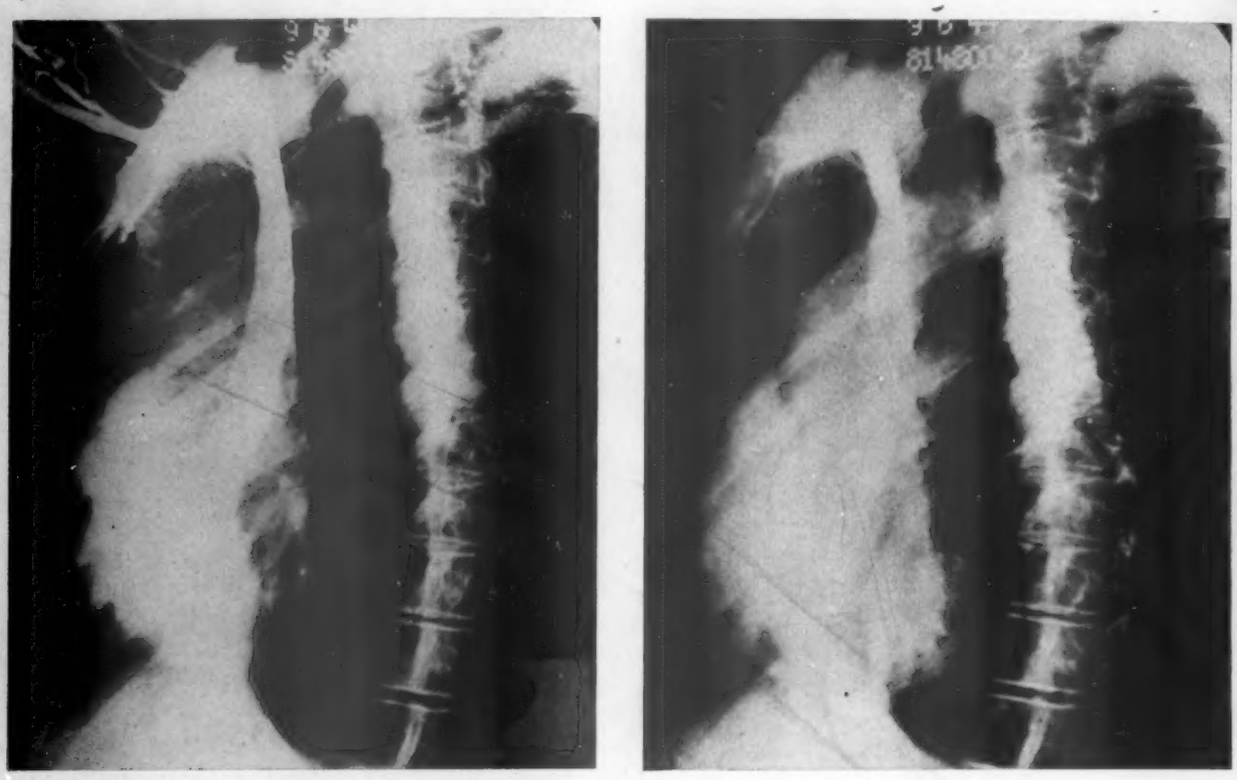


FIGURE 4. *Diagrammatic Representation of the Heart in Tetralogy of Fallot, with the Outer Wall of the Right Ventricle Removed.*

*Note the stenosis of the pulmonary artery, interventricular communication, deviation of the aortic orifice to the right and hypertrophy of right ventricle.*

lies not with Fallot,[134] but with his translators, for he was perfectly clear in describing: "stenosis of the pulmonary artery, interventricular communication, deviation of the origin of the aorta to the right, and hypertrophy, almost always concentric, of the right ventricle" (Fig. 4). Thus, all cases of *la maladie bleue* are associated with a dextroposition of the aortic orifice; only 20 per cent of these patients have a right aortic arch and descending aorta. (This is the usual occurrence in birds,[135] and might be referred to as the "bird aorta.")



A B

FIGURE 5. *Unretouched Angiocardiogram in Tetralogy of Fallot.*

A. *Five seconds after injection of the dye, which is concentrated in the superior vena cava and right side of the heart.* B. *Two and a half seconds later (or the same time as that in Figure 1) instead of entering the pulmonary circulation, most of the flow is shunted through the displaced aortic orifice into the systemic circulation; a small amount is entering the stenotic pulmonary artery.*

Although simple defect of the ventricular septum (*maladie de Roger*) is a relatively common congenital anomaly and can be well tolerated, its association with hypoplasia of the pulmonary artery results in a serious clinical syndrome. Instead of the blood flowing through the defect from the left ventricle to the right ventricle, the stenosis obstructs the pulmonary circulation sufficiently to reverse the direction of the flow in the ventricles. Aeration of the resultant small amount of circulating blood in the pulmonary circuit is not sufficient to prevent cyanosis, which occurs in the presence of 5 gm. of reduced hemoglobin per 100 cc. of blood.[136] The value for the total hemoglobin may become 26 gm. per 100 cc., and the red-cell count may become as high as 12,000,000.[137]

It was formerly assumed that all the blood that passed through the lungs was not fully oxygenated, so that most investigators believed that a further increase in the circulation of the blood to the lungs would be of no benefit. However, it was observed that the condition of children with pulmonary stenosis and associated patent ductus arteriosus became worse if the duct was closed, thereby reducing further the flow of blood to the lungs. Consequently, Taussig[62] proposed that these patients might be improved if a means could be devised whereby a greater volume of blood could reach the lungs.

On the basis of this suggestion, Blalock[132] devised an operation in which a systemic artery was anastomosed to one of the pulmonary arteries and thus increased the pulmonary flow. Doyen[54] was the pioneer in this particular location, having attempted in 1913 to divide what was believed to be a stenotic valve with a tenotome knife. The patient died several hours later, and examination revealed the usual finding — namely, that the defect is commonly in the pulmonary conus rather than in the

valve itself. (Recent work of Brock is discussed below.)

In untreated cases of pulmonary stenosis the life expectancy is only twelve and a half years, with a maximum of about twenty-five years. Chief complications are cardiac failure, poor development, pulmonary tuberculosis, cerebral thrombosis and bacterial endarteritis.

The underlying principle in the choice of patients for operation is that there must be an inadequate flow of blood to the lungs. The two outstanding diagnostic features are roentgenographic evidence that the pulmonary artery is small and clinical and roentgenographic evidence of absence of congestion in the lung fields.

Additional information concerning the pathologic physiology of congenital heart disease has been supplied by Bing et al.[138] and by Dexter and his group.[88] In employing the method of intravenous catheterization for studying cardiac dynamics, Bing, Vandam and Gray[138] have been able to measure the outputs and pressures of the various chambers of the heart, the pulmonary artery and the total pulmonary flow, including that through the collateral channels. This has enabled them to make exact differential diagnoses in difficult cases. Bing states that the three most important indications for surgical creation of an artificial ductus arteriosus are: a decrease in effective pulmonary blood flow; a reduction in pulmonary-artery pressure; and a diagnosis of intracardiac shunt. The limitations and potentialities of such methods have been described by Dexter et al.[88] Auricular and ventricular septal defects offer greater difficulty in diagnosis. Also of distinct value is visualization of the chambers of the heart and great vessels[139] (Fig. 5) — a procedure just being adopted for routine use in many centers.[140]

The preferred age for operation is from five to nine years, since the infant mortality is approximately 30 per cent as compared to one of 10 per cent for children.[141] Taussig[142] does not advise operation in those eighteen months and younger, unless the infant is doing very poorly and it appears that the chances of survival to an old age are less than 50 per cent. The early development of attacks of paroxysmal dyspnea is an unfavorable sign. The period of greatest difficulty occurs when the ductus arteriosus is being obliterated — usually between six and eighteen months. As collateral circulation and compensatory polycythemia develop (one to three years) attacks of dyspnea diminish. Most infants who survive until eighteen months of age manage to hold their own for a time, and then begin to improve.

Leeds[143] showed in dogs that anastomosis of the aorta to the pulmonary artery could be done. Utilizing this approach and by means of an ingenious clamp, which, although reducing the aortic lumen, still allows approximately 50 per cent flow during its application, Potts et al.[144, 145] are doing aortic-pulmonary anastomoses. Advantages are that the shunt can be made whatever size is deemed necessary, and that the innominate artery can be conserved in younger patients. At last report they have operated on 41 patients for pulmonary stenosis, 10 below the age of two and three below one year of age. Potts reports 4 deaths in this series.

The anastomosis of the left subclavian artery to the left pulmonary artery is advocated by Holman.[146] Because of sharp angulation of the subclavian artery in 4 cases, he has deliberately divided the left pulmonary artery proximal to the anastomosis, and has noted no subsequent detrimental effects.

Blalock[147] still prefers an end-to-side anastomosis, using the subclavian artery in most patients who are more than two years of age. The desired aim is to choose a systemic artery of such caliber that the polycythemia and cyanosis will disappear but not large enough to place undue strain on the heart. The incision in the chest is usually made on the side opposite to that which the aorta descends (as mentioned above). It is desirable to use the subclavian branch of the innominate rather than the opposite subclavian, which arises directly from the aorta, since the former makes a less acute angle with its parent artery after the anastomosis is performed.

Preoperative studies will have given a good indication of the size of the artery required — the greater the degree of arterial oxygen unsaturation, the larger the vessel that is needed. It has been found that the pressure in the pulmonary artery as determined at the time of operation is generally equivalent to 150 to 240 mm. of water. If the pressure is elevated in excess of 300 mm. approaching or equaling the aortic pressure, the chances are that there is an absence of the ventricular septum, and the procedure should be canceled since the flow of blood inevitably depends upon the relative pressure between the systemic and pulmonary circulations.[147]

If the patient has pronounced polycythemia and if the loss of blood during the operation is minimal, whole blood equal to approximately 1 per cent of the body weight is removed at the conclusion of the operation. It has been found that there need be no concern about the circulation of the arm when the subclavian artery is used; however, when the innominate artery is divided there is danger of cerebral ischemia or thrombosis.

Up to the present time Blalock and his associates have operated upon 415 patients, who were thought to have tetralogy of Fallot. The over-all mortality, including deaths among those in whom an anastomosis could not be performed, those in whom the diagnosis was in error and those who have died since leaving the hospital, has been reduced to 18 per cent. In 283 cases in which the subclavian

artery was used for the shunt the mortality was 10 per cent. On the other hand, when the carotid or innominate artery was utilized,[132] the rate was approximately 30 per cent. It should be emphasized that surgery is not indicated for cor biloculare or cor triloculare, persistent truncus arteriosus and Eisenmenger's complex — distinguished from the tetralogy of Fallot by a normal or even dilated pulmonary artery.

### Recent Developments

Some of the remaining anomalies are being ameliorated or remedied by surgical procedures. Others are in the process of investigation in the experimental laboratory.

Gross,[148] in 1945, reported the first successful surgical division of a congenital vascular ring around the trachea.

Tricuspid atresia, until recently considered inoperable,[149] has now come to surgery. Potts and Gibson[150] described 3 patients successfully operated upon with performance of aortic-pulmonary anastomosis. The physical findings in this anomaly are identical with those encountered in the tetralogy of Fallot. Differential features are left-axis deviation in the electrocardiogram (the only lesion that causes cyanosis and has this characteristic) and hypertrophy of the left ventricle and hypoplasia of the right ventricle, which produces a suggestive roentgenographic silhouette.

Shumacker[151] has recorded the first case of coarctation of the aorta complicated by a saccular aneurysm just distal to the stenosis successfully treated (February, 1947) by resection of the stenotic area and aneurysm with repair of the aorta by end-to-end anastomosis. Alexander and Byron[152] had a similar case, which was successfully treated by resection alone without anastomosis.

Brock[153] has recently recorded his experiences with the direct approach of Doyen to the pulmonary valve in cases of pulmonary stenosis. This method was restudied because of his belief that the stenosis is purely valvular or diaphragmatic in a proportion of cases far higher than heretofore supposed. He examined the valves, at operation, by means of a cardioscope inserted through the pulmonary artery, but has decided that this approach is unsatisfactory.

The first patient was operated on in February, 1948. A valvulotome was inserted through an incision in the wall of the right ventricle and used to divide the pulmonary valves. A special dilating forceps was then passed and opened fully in the region of the stenosis. The patient, an eighteen-year-old girl, survived and showed decided improvement subsequently. The procedure has been repeated in 2 more cases, in both of which the patients survived. This operation is not meant to supplant the Blalock procedure, but it does have definite advantages in cases in which the stenosis involves the pulmonary valve itself rather than the pulmonary conus. The exact proportion of such cases will have to be reinvestigated, but this work of Brock's represents another pace-setting advance. (In 155 cases of pulmonary tract anomalies studied by Keith,[154] a defect was found in the pulmonary conus in 133, and in the pulmonary valve alone in 22; he estimated that the pulmonary conus would be involved in 90 per cent of the cases.)

Hanlon and Blalock[155] have recently shown experimentally that anastomosis of the pulmonary vein to the superior vena cava can be done. If feasible in man, this delicate procedure offers a possible approach to the surgical treatment of complete transposition of the aorta and pulmonary artery.

Utilizing the same approach and occlusive clamp, but shifting the operative field slightly distalward, the same workers[156] have been able to obtain an entirely different result — that is, the production of an interatrial septal defect under direct vision, without interruption of the circulation, with minimal loss of blood and with fairly accurate control of size — truly a brilliant achievement. Though he suggested a blind approach through the jugular vein, Jarotzky[157] apparently was the first to recommend such a shunt, knowing that the patient with a mitral stenosis associated with an open foramen ovale (Lutembacher[158] syndrome) has a comparatively good prognosis (forty years[159]).

Gibbon and Templeton[160] have recently reconstructed and replaced leaflets of the tricuspid valves with pericardial and venous grafts in dogs.

In the field of myocardial ischemia Beck[161] has continued pre-eminent in the search for methods to revascularize the heart. Louis Gross[162, 163] made the important discovery that ligation of the coronary sinus produced extensive and abundant dilatation of the intramyocardial collateral channels. Sutton and Lueth[164] found that the pain fibers involved in experimental coronary occlusion are located in the adventitia of the coronary artery. Fauteux[165] combined these last two facts by ligating the magna cordis vein alone (more distal than the coronary sinus) or combined with pericoronary stripping in a total of 15 patients over a six-year period. His results are encouraging.

In view of these advances J. T. Roberts[166] (another internist), suggested to Beck[161] the possibility of making an arteriovenous fistula to the heart to increase a deficient blood supply. Utilizing various cannulae[166] as well as the Potts clamp, Beck and his workers have been successful in grafting a systemic artery into the coronary sinus in dogs. They have recently reported the first case (January, 1948) in a human being in which a new connection was made from the aorta to the heart with a transplanted segment of brachial artery. The patient recovered from this extraordinary operation.

Gross and his associates[167] report encouraging early postoperative results after the use of arterial

transplants in the treatment of cardiovascular defects.

Polished tubes of a plastic (methyl methacrylate) material containing a movable valve were inserted in the aorta by Campbell[168] in dogs with surgically produced aortic valvular insufficiency. Clotting of blood about the moving valve did not occur for the length of time the animals were observed. The clinical use of such materials, particularly polythene, has a promising future in surgery.[169, 170]

Smithy et al.[171, 172] are reinvestigating the use of the valvulotome as applied to aortic stenosis. The instrument was first introduced through the aorta, but later work has shown that the wall of the left ventricle offers the most effective approach. This approach was also found best by both Cushing[24] and Bernheim[25] forty years ago. In those days MacCallum[23] and Allen[32] were introducing their valvulotomes and cardioscopes through the left auricular appendage. Cushing reported the first successful attempt to produce valvular lesions of the heart by an intrathoracic approach. He emphasized then that a successful operation must be done on the animal before being attempted on man.

* * *

Some understanding of the surgical approach to cardiovascular disease has become mandatory for the present-day clinician. The recent advances in treatment of pulmonary stenosis, coarctation of the aorta, anomalies of the aortic arch and patent ductus arteriosus are brilliant and promising refinements in the art of surgery. In fact, their diagnosis and treatment are now on a highly scientific plane. The operative treatment of acquired cardiac disease has been reintroduced. Further technical advances are bound to occur. Such intricate and involved procedures call for highly trained and skilled groups of workers — cardiologists, pediatricians, surgeons, anesthetists, roentgenologists, physiologists and probably others. Whether such procedures can or should be done by the average well trained surgeon in the average hospital is doubtful. On the other hand, whether it is economically or socially wise for the patients requiring these procedures to go to special regional centers is also a moot question. Physicians and surgeons should lead the way not only in introducing these new methods but also in discussing and suggesting means to assure their availability to all groups in all regions of the country.

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# CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

## Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

## CASE 35011*

### PRESENTATION OF CASE

A twenty-two-year-old woman was admitted to the hospital complaining of pain in the left leg.

Ten years before admission, following a fall, she had pain in the lower back for several months. Six weeks before admission she began to notice pain along the anterior aspect of the proximal third of the left lower leg, deep within the muscle group. This leg pain was "wave-like, deep and boring." It was fairly constant and more marked after exercise, but not incapacitating. It did not keep her from sleeping, except to waken her early in the morning. The pain sometimes radiated up the posterior thigh and lower back but never farther distally than the mid-lower leg. In the week before admission she noticed weakness of the left foot when she was tired.

On physical examination the left calf was 0.6 cm. larger than the right, and there was questionable prominence of the head of the left fibula. There was no calf tenderness or sensory change. The muscle power in the foot and ankle was equal bilaterally. There was slight tenderness in the left buttock. The ankle jerks were equal; the knee jerk seemed to be more active on the left.

The blood pressure was 140 systolic, 92 diastolic.

Examination of the blood demonstrated a normal white-cell count, hemoglobin and urine. The sedimentation rate was 4 mm. in one hour. The blood calcium was 9.8 mg., and the phosphorus 4.1 mg. per 100 cc., and the alkaline phosphatase 2.8 units.

X-ray examination of the skull, lumbar spine and pelvis was not remarkable. There was no evidence of bone destruction or alteration of the intervertebral spaces. Examination of the left knee showed no joint abnormalities. There was a small radiolucent area 7 mm. wide and 15 mm. long in the medulla of the proximal third of the fibula. There were one or two small, irregular septums within the lesion, the margin of which was smooth and slightly sclerotic. The overlying cortex was slightly elevated, but there was no break in the cortex and no periosteal reaction (Fig. 1).

An operation was performed.

### DIFFERENTIAL DIAGNOSIS

DR. WILLIAM ELLIOT†: I think we can rule out anything in the spine because of the absence of sensory changes in the leg, muscle weakness and the fact that there is no atrophy. As far as the skull is concerned, I see nothing there that is of interest, and I see nothing remarkable in the dorsal spine.

In consideration of a lesion in the bone we either consciously or subconsciously decide that it is infection or a tumor. In this case I believe we can more or less rule out an infectious process; certainly, it was not an acute infectious process. If it was a tumor, it forces us into a large group of possibilities, and first we must decide whether it was malignant or benign. There is nothing to indicate that this

FIGURE 1.

was a malignant tumor. There was no new-bone formation, periosteal proliferation or any of the other stigmas we expect to see in malignant tumor.

As to the other conditions that this may represent, I think we can rule out hyperparathyroidism on the blood chemical findings as reported here. In most of the lesions of hyperparathyroidism that we have encountered there has not been an increase in thickness of the cortex over the lesion. Usually the cortex has been thinned out, and this is also the x-ray finding in fibrous dysplasia and certain

*Presented at a meeting of the New England Cancer Society held at the Massachusetts General Hospital.

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other lesions. As I said before, this apparent thickening of the cortex at the site of the lesion is important. There is a condition described by Jaffe and Lichtenstein[1] as nonosteogenic fibroma, which is benign and, to my knowledge, gives no symptoms. It may occur along the shaft of the long bones, and in a case that we have followed over a period of years there was definite thinning of the cortex, but no symptoms referable to the site of the lesion. The nonosteogenic fibromas produce no symptoms and, as a rule, are incidental findings.

Another possibility is a cartilage inclusion, which occurred at the time the line of the epiphysis was at the site of the present lesion, and moved down the shaft with the increase in the length of the bone. Nothing is said about point tenderness — a finding usually present in osteoid osteoma. Whether or not osteoid osteoma is a clinical entity or the result of low-grade infection I will not discuss at this time. I believe this was an osteoid osteoma, and I base this opinion on the fact that these lesions may occur in the medullary cavity against the inner surface of the cortex or within the cortex itself. Pain is the symptom that causes these patients to seek medical advice. Pain, as a rule, is slow in onset; it becomes more aggravated as time goes on, and it may be quite constant. The physical findings are more or less absent except for point tenderness over the lesion itself. Or there may be slight swelling about the lesions, which this patient had. I do not believe this was eosinophilic granuloma because again there is usually bone destruction and a little more reaction about the lesion itself in that condition.

Regarding the operation that this patient had, I believe it was a simple excision of the lesion, with possible preservation of the cortex away from the lesion.

Dr. Grantley W. Taylor: I wonder why Dr. Elliot did not allude to benign giant-cell tumor in that area.

Dr. Elliot: I should have. Benign giant-cell tumor does occur along the shaft. A giant-cell tumor of the size of this lesion would have been asymptomatic, and again there is thinning of the cortex as a rule at the site of the tumor. This cortex was a little elevated and thickened, which I believe is against benign giant-cell tumor.

Dr. James W. Jameson: Do you place any significance on the pain occurring at night?

Dr. Elliot: That frequently happens in cases of osteoid osteoma.

Dr. Philip Batchelder: Does it have a nidus in the center?

Dr. Elliot: There is a small area of increased density in the cortex, but I cannot be sure that this represents the nidus. I believe Jaffe[2] states that the nidus may disappear as the lesion matures.

Dr. George C. Levene: I would agree that from the description this was osteoid osteoma.

Dr. Douglas J. Roberts: Would you consider fibrous dysplasia?

Dr. Elliot: I had considered that. Quite frequently the cortex shows a little thickening and may be bulging, of course; but usually there is a little thinning and not the elevation that this patient had. I think you can see on the films a definite thickening of the cortex as it comes up along there, which I believe is the sclerosis that has been described in the osteoid osteoma.

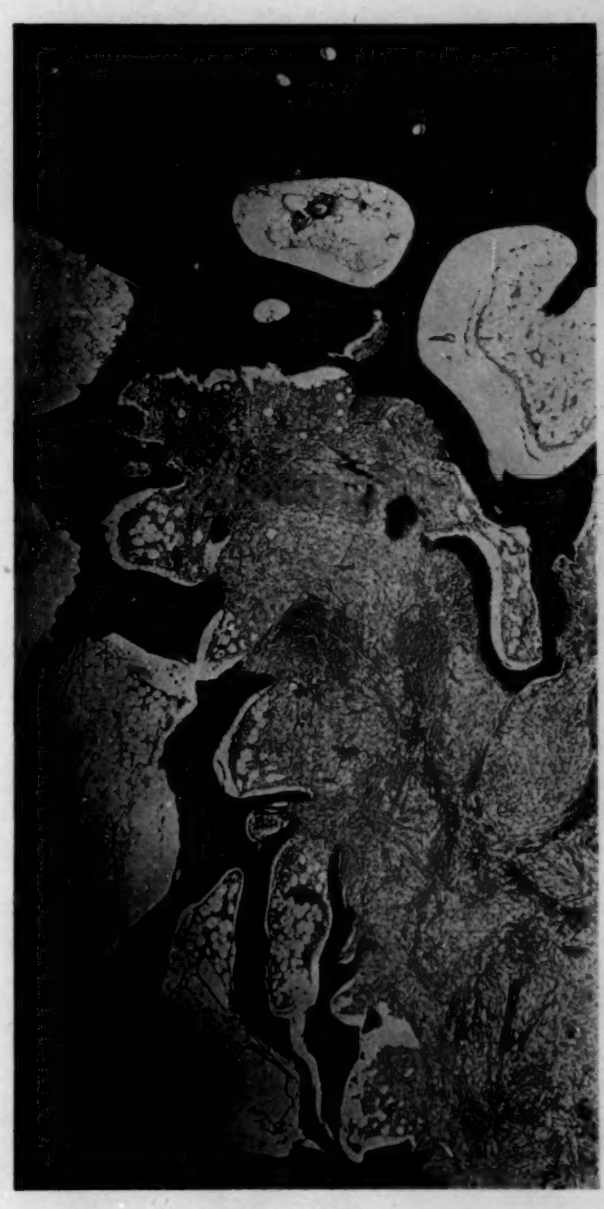

Figure 2.

### Clinical Diagnosis

Benign tumor.

### Dr. Elliot's Diagnosis

Osteoid osteoma.

### Anatomical Diagnosis

*Nonosteogenic fibroma of bone (fibula).*

### Pathological Discussion

Dr. Benjamin Castleman: This patient was operated on by Dr. Edwin Cave, who resected the lesion in toto. I have some microscopical sections of it, which I will show. The first slide (Fig. 2) is a low-power view of the lesion showing cortical

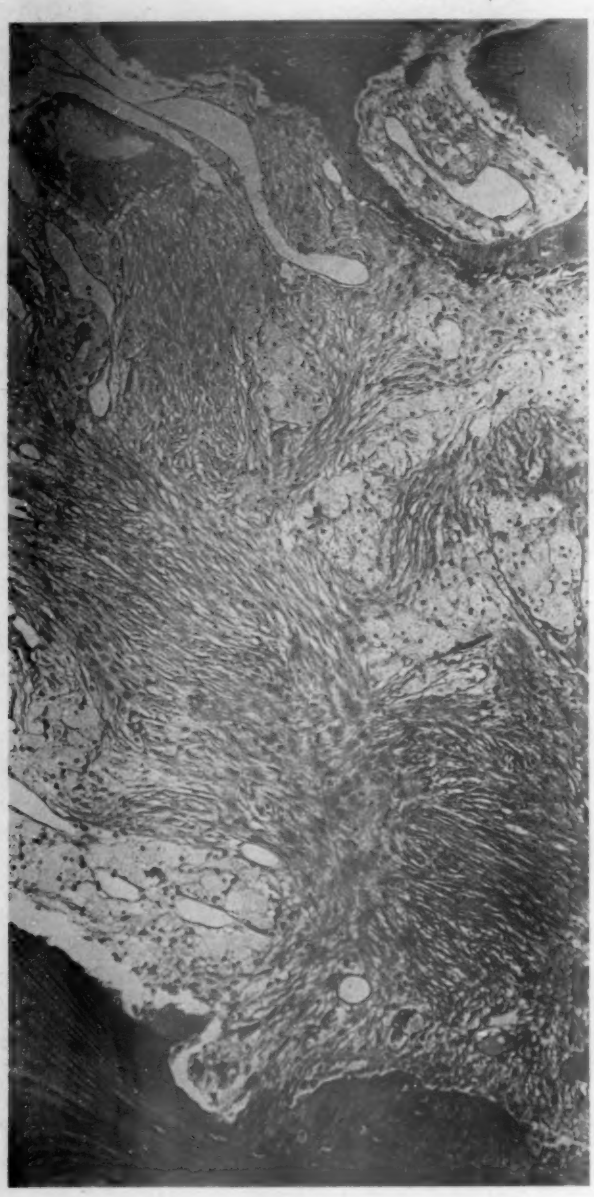

Figure 3.

bone at the top and a loose fibrous lesion in the medulla. Note the normal fatty marrow around part of the lesion. A higher magnification (Fig. 3) shows that the lesion is made up almost entirely of thin, spindle-shaped fibroblasts without any evidence of osteogenesis. In addition, there are a great many cells filled with fat, which are undoubtedly merely phagocytes. The whole picture fits in exactly with what Jaffe[1] called nonosteogenic fibroma. It is about the second or third case that we have seen here. These tumors, according to Jaffe, do produce occasional symptoms. I do not believe they are all symptomless. They often cause pain and almost always occur in children between the ages of eight and sixteen, usually under twenty in contrast to giant-cell tumor, which occurs almost always above twenty. It is a tumor that in the past has been called a variant of giant-cell tumor. It certainly does not look at all like osteoid osteoma. The lesion is perfectly benign.

Dr. Elliot: I thought that the lesions were always incidental findings.

Dr. Castleman: In his report of 10 cases Jaffe stated that in half there was a history of trauma that apparently attracted attention to the area where the lesion was found by x-ray examination.

Dr. Clinton Mullins: How can you differentiate that from a healed osteomyelitis?

Dr. Castleman: There is no evidence of inflammation at all, and the type of connective-tissue cell is very dense, without any cellular reaction.

### References

1. Jaffe, H. L., and Lichtenstein, L. Non-osteogenic fibroma of bone. *Am. J. Path.* **18**:205-221, 1942.
2. Jaffe, H. L. Osteoid-osteoma of bone. *Radiology* **45**:319-334, 1945.

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## CASE 35012

### Presentation of Case

A forty-eight-year-old carpenter was transferred from another hospital because of an abdominal-incision dehiscence and draining small-bowel fistula.

The patient had been well and working up until three months before entry, when he began to feel dull, "crampy" pains in the epigastrium, lasting a few minutes. These occurred several times a day and were relieved by two or three glasses of whisky. They were not related to meals. The patient stated that for many months he had often had a desire to defecate without success. Five weeks before admission the pains became more severe and frequent. He stopped work and started drinking as much as a pint of whisky each morning, with little relief of pain but with nausea and vomiting of gastric contents. One week later the pain suddenly became very severe after he had taken a large glass of whisky. He became "pale and dizzy," and his upper abdomen "very hard." He was very weak and drove his car home with great difficulty. His abdomen was painful to touch. The following day he was admitted to another hospital. Physical examination revealed an obese, middle-aged man complaining of upper abdominal pain. His face was flushed and he perspired profusely. His temperature and pulse were normal. The chest was

clear and resonant throughout. The upper part of the abdomen was distended, firm and very tender. The lower part of the abdomen was much softer and not distended. The abdomen was tympanitic, and peristaltic movements could not be heard. Repeated enemas gave no results. In the other hospital the patient's temperature remained at 99°F., and the pulse was normal for three days. The distention became greater, and the pain generalized. There was difficulty in breathing. He at no time vomited or felt like vomiting. A Wangensteen tube was introduced, and a large amount of fluid recovered in a twenty-four-hour period. The following day his condition was worse. A diagnosis of retrocecal appendix was made, and a laparotomy done. The large bowel was distended. The intestine showed acute enteritis bordering on a purple color. The appendix was perfectly normal and was removed. A colostomy was performed to relieve the distention. The distention persisted. Three days later a fecal fistula formed about 8 cm. above the colostomy opening. There was induration below the colostomy, with questionable abscess formation. Two days later the abscess was drained, and the patient improved. His appetite was better. After a period of two weeks there seemed to be a breaking down of the incision, owing to the fecal fistula and colostomy. The patient was given penicillin and intravenous fluids. Following this he developed dyspnea and marked perspiration. Intravenous fluids were cut down to 500 cc. three times a day. He had been taking moderate amounts of fluid and was co-operative at all times. He grew progressively weaker. At no time following operation was there any bowel movement, although repeated enemas were tried. The abdomen remained distended and tympanitic. The wound drained constantly and showed no attempt to close. The urine showed a ++++ test for albumin, and the sediment contained casts and a few red and white cells. The blood hemoglobin was 90 per cent and the red-cell count 4,590,000. The white-cell count was 7800. Two days after admission the blood sugar was 136, the nonprotein nitrogen 60 and the creatinine 29 mg. per 100 cc. Twenty-four hours after operation the nonprotein nitrogen was 39 mg. per 100 cc., and on the following day it was 35 mg. per 100 cc. The white-cell count was 22,000. Six days later it was 36,200. On the thirty-third hospital day he was transferred to this hospital.

Physical examination showed the patient to be oriented and in only slight distress. The skin was warm and moist. The tongue was red and heavily coated. There was some bleeding about the gums. There were dullness and many bubbling rales at the right base. The abdomen showed slight tenderness. Peristalsis was normal. On the right side of the abdomen there was a large wound dehiscence, with viable bowel lateral to the rectus muscle. Two stomas, one of the ileum and another of the colon, were thought to be identified. There was extensive undermining into the flank, with draining sinuses. The rectum was filled with barium.

The temperature was 100°F., and the pulse 92. The blood pressure was 95 systolic, 70 diastolic.

A review of the symptoms revealed that the patient had been accustomed to drinking a half to two bottles of beer a day for many years and that during the present illness he had drunk up to a pint of whisky a day. He had had no previous operations or serious illnesses. He usually had two or three bowel movements daily.

Two days after admission laboratory study revealed a serum nonprotein nitrogen of 35 mg. per 100 cc., and the total protein was 6.41 gm., the albumin 3.85 gm., and the globulin 2.56 gm. per 100 cc. (albumin-globulin ratio, 1:5). The serum chloride was 84, the sodium 119.6, and the potassium 5.6 milliequiv. per liter. The cephalin flocculation test was negative in twenty-four hours and ± in forty-eight hours. The van den Bergh reaction was normal. The white-cell count was 23,200; the blood hemoglobin was 10.7 gm. The urine was negative for albumin. The prothrombin time was 19 seconds (normal, 16 seconds).

Roentgenograms of the chest and abdomen taken on the second hospital day showed plate-like atelectasis at the left base and some fluid in the left pleural cavity. There was no distention of the large or small bowel. There were remains of barium in the transverse colon and rectum. There was no definite evidence of free air beneath the diaphragm.

In the hospital the patient's condition became progressively worse. The chest became full of rales, and respiration was difficult. On the fourth hospital day the temperature suddenly rose to 103°F., and the white-cell count to 36,400. The abdomen was somewhat distended, although there were large quantities of fecal drainage. The following day a Harris tube, which had been passed the day before in preparation for a proposed ileotransverse colostomy, was found coiled in the stomach and withdrawn. Nearly all the gas had disappeared, and there appeared to be less abdominal distention. The next day a barium enema showed no obstruction as far as the hepatic flexure. The cecum and ascending colon could not be outlined. Examination of the blood at that time revealed a sodium of 130.7, chloride of 91, potassium of 3.3 and carbon dioxide of 30.5 milliequiv. per liter. The fasting blood sugar was 87 mg. per 100 cc. The white-cell count was 18,500, with 77 per cent neutrophils.

On the seventh hospital day the patient developed symptoms suggestive of delirium tremens with "shakes" and hallucinations. He became semi-delirious and was incontinent of urine. The pulmonary situation became critical, with progressive respiratory embarrassment. There were coarse

raies and rhonchi over the entire chest. Repeated catheter suctioning of the tracheobronchial tree was productive of a large amount of thick, stringy, whitish, tenacious mucus.

On the tenth hospital day the temperature had risen to 105°F. The total protein was 5.8 gm. per 100 cc., the amylase 22 units per 100 cc., and the carbon dioxide 26.9 and the chloride 100 milliequiv. per liter. The calcium was 7.8, and the phosphorus 3.4 mg. per 100 cc. The urine output dropped to 750 cc., and the nonprotein nitrogen rose to 60 mg. per 100 cc. The patient was flushed and mildly cyanotic. The blood pressure was unobtainable. The pulse rate was 150, and the respirations 44.

The following morning, on the twelfth hospital day, the temperature had risen to 106°F., shortly after which the patient died.

## Differential Diagnosis

Dr. Marshall K. Bartlett: May we see the x-ray films?

Dr. Stanley M. Wyman: The original films of the chest show the left leaf of the diaphragm elevated, and there is horizontal linear density, which appears to lie chiefly in the region of the left lower lobe. There is no definite or significant fluid made out in the pleural space in the lateral view, but there is a suggestion of some blunting of the costophrenic angle in the posteroanterior view. The same day an upright film of the abdomen shows a gas-filled stomach at a considerable distance from what I take to be the diaphragm, and there is some suggestion of mottled rarefaction adjacent to the undersurface of the diaphragm. This is not definite but suggests gas in the space below the left leaf of the diaphragm. A film taken four days later shows essentially the same process, with atelectasis of the left lower lobe and a little more suggestion of fluid in the left costophrenic sinus. A film taken still later, at the time of barium-enema examination, shows no definite intrinsic lesion in the colon, but it does show a coiled tube in the stomach. Again, there is a suggestion of mottled rarefaction adjacent to the left leaf of the diaphragm, possibly lying below it. The last film of the chest shows essentially a progressive change, with atelectasis and probably fluid.

Dr. Bartlett: Let us approach this problem by going back to the first admission to the outside hospital and see what the history seemed to be then and, putting that together with the operative findings, see if we can figure out any condition that will fit this picture. In the first hospital admission there was a history, going back approximately two months, of dull, crampy, epigastric pain, lasting a few minutes, occurring several times a day and relieved by two or three glasses of whisky. Then we have the statement that for many months he had a desire to defecate without success. Later in the history we are told that the normal bowel movements were two or three a day. This may have represented a change in bowel habit. He went along like that until about a week before admission, when the pain became more severe, and he began drinking more to relieve it, without success. At that time he had nausea and vomiting of gastric contents. That is the only time in the story that he had nausea and vomiting. On admission the striking thing in the physical examination seemed to be that the abdomen was distended, the upper portion more so than the lower portion. For the first three days in the hospital he had a temperature of 99°F. and a normal pulse. The white-cell count was normal until after operation, and then it rose to more than 20,000. At operation a normal appendix was found. However, the large bowel was distended, and a note was made that "the intestine showed acute enteritis." It is not specified whether this was the large or the small bowel. I assume, since it is a separate statement, that it refers to the small bowel. Its color was purple. What condition could produce that picture? The changes in bowel habit and the facts that at operation the colon was distended and that on barium enema the barium did not pass the hepatic flexure certainly bring to mind the possibility of an obstructing lesion in the large bowel. On the other hand, there are a good many things that seem hard to explain with that diagnosis. The location of pain during the two months before admission was epigastric. Certainly, lesions of the colon usually produce pain at a lower level than that. We are all probably familiar with the experimental work that Dr. Chester M. Jones did some years ago by distending balloons at various levels of the gastrointestinal tract to see where pain was produced in the abdomen. Distention of the hepatic or splenic flexure might produce pain in the right or left upper quadrant, but this epigastric pain is hard to explain on the basis of a lesion in the colon. It is also hard on that basis to explain the fact that the distention was limited to the upper abdomen, and the colostomy does not seem to have done any good, so I do not believe that an obstructing lesion of the colon fits the picture very well.

What lesion of the small bowel could he have had? Could it have been a tumor? A lesion like that should express itself either by obstruction or by bleeding. We have no evidence here that he had massive bleeding into the gastrointestinal tract. Could he have had an obstructing lesion in the small bowel? I think it would be very unlikely to have such a sudden change in the picture, with his mild symptoms becoming quite suddenly worse, unless we assume that the lesion perforated, but if such were the case, the findings at laparotomy would have been different. If he had a small-bowel obstruction, we would not expect the colon to be dilated and we would expect that both dilated and collapsed loops of small bowel would be found. So I do not believe that fits the picture very well.

It is noted that the bowel was almost purple. That suggests interference with blood supply. Could it have been a mesenteric thrombosis? I think that is unusual with such a gradual onset. Mesenteric thrombosis should have been a more acute picture, and I doubt if the outcome would have been so long drawn out.

Could this have been regional enteritis? Of course, the thing that brings that to mind is the fact that he developed a spontaneous small-bowel fistula where the colostomy was done, and since regional enteritis is notorious for forming fistulas, we must consider it. So far as the history is concerned, any change in the bowel habit has been in the direction of constipation rather than toward diarrhea. I think the pain could be consistent with an inflammatory lesion in the small bowel, but I rather expect that the description of the findings would be different at operation in that event. As we all know, regional enteritis is typically a fairly circumscribed or localized lesion. There may be multiple areas, but usually they do not cover a great extent of bowel in any one area. However, it is a possibility. I do not see how we can rule it out. Again, the distention, if due to a small-bowel lesion, would probably have been generalized and not limited, as this was, to the upper abdomen. Perhaps I am putting too much stress on that finding, but certainly it is emphasized in the history.

To move up the gastrointestinal tract — could this have been gall-bladder disease? It would be very atypical, and I do not believe the evidence fits the picture particularly well. It would not account for the changes in the small bowel at operation. The distention of the colon, perhaps, could have been purely secondary — that is, paralytic. Could this have been pancreatitis? I do not see how one can rule it out. It is unusual for pancreatitis to cause mild symptoms for a period of two months and then an acute attack. I would like that possibility better if this had come on suddenly, out of a clear sky. Pancreatitis typically occurs after a large meal or the ingestion of a considerable amount of liquor. This man drank quite a lot, but the onset was not typical. The normal amylase taken a month after the onset of the attack does not rule pancreatitis out at all. Typically, the amylase goes up in the first twenty-four or forty-eight hours and then comes down to normal. It could have come down long before this determination was made and still that diagnosis could be the correct one.

How about acute gastritis? I should think it could produce the symptoms that he had up to the day he came into the hospital, — mild, dull, crampy, epigastric pain, — but it is hard to explain the development of acute, severe pain on the basis of simple gastritis.

How about an ulcer of the stomach or duodenum or possibly a gastric neoplasm that had perforated? I believe the history of the symptoms for two months before entry fits that reasonably well. I think the increase in pain for a week before admission could be explained on the basis of gastritis, the sudden, severe pain the day before admission representing a perforation. If we assume that it was one of those rare situations in which the perforation was into the lesser peritoneal cavity, which then became distended with air and fluid, we perhaps can explain the difference between the upper and lower abdominal distention. Could that explain the operative findings? If we do assume that he had perforation in the upper abdomen, even in the lesser peritoneal cavity, there probably would have been some leakage of irritating fluid into the general peritoneal cavity, resulting in generalized peritonitis, which might have caused the changes noted in the bowel. Distention of the large bowel we would assume on the basis of paralytic ileus, and the fact that colostomy did not work reinforces that thought. Was this an ulcer or a neoplasm? I cannot see any way to be sure of either. The patient was forty-eight and had a short history. It seems on a chance that it is more likely that he had ulcer than neoplasm, with perforation.

Accepting the situation as it was at operation, what happened from there on? It seems to me that it is all relatively simple: a small-bowel fistula developed. Why it developed I cannot be sure. If he had regional enteritis, that is reason enough in itself, or it is possible that in closing the wound in a distended patient, some injury was done to the bowel, which in the course of several days could have resulted in perforation. He developed a small-bowel fistula, wound sepsis, a great deal of loss of fluid and electrolytes from the small-bowel fistula, resulting in the imbalance in the blood chemistry that is outlined. Also, there seems to have been very little question that he had a septic process in the left upper quadrant under the diaphragm. There

is certainly a strong suggestion in the x-ray picture.

A barium enema was then done. If this patient had a lesion of the upper abdomen, why did the barium enema fail to fill the right colon? I cannot answer that. I do not know whether it is safe to rule out an organic lesion in the hepatic flexure. I am inclined to do so and to say that this was something high up in the abdomen. The course was steadily downhill — a losing struggle against sepsis and a small-bowel fistula. The downhill course continued with progressive pulmonary difficulties. There was a thought at one time that ileotransverse colostomy could be done. That was probably on the basis of a rather desperate attempt to correct the fluid and electrolyte loss from the small bowel by short-circuiting the fistula and the colostomy. The patient was apparently not well enough to allow that to be done and went rapidly downhill and died. It seems that the downhill course was a mixture of sepsis, fluid and electrolyte loss, and pulmonary difficulties, with probably terminal bronchopneumonia.

### Clinical Diagnoses

Wound dehiscence, with small-bowel fistulas.
Chronic alcoholism, with delirium tremens.
Bronchopneumonia.

### Dr. Bartlett's Diagnoses

Gastric ulcer, with perforation.
Small-bowel fistulas.
Bronchopneumonia.

### Anatomical Diagnoses

*Acute pancreatitis, with perforation of transverse colon and abscess formation.*
Peritoneal abscesses, with fat necrosis.
Small-bowel fistulas.
Operation: colostomy.
Pulmonary atelectasis.
Pulmonary edema.
Hepatomegaly, with fatty vacuolization.
Acute splenitis with splenomegaly.

### Pathological Discussion

Dr. Benjamin Castleman: At autopsy there was, as to be expected, a lot of sepsis around the two colostomies that were present in the right lower abdomen. When the abdominal cavity was opened there was a gush of gas; the omentum was plastered down over the peritoneum and was thick and porky, suggesting long-standing sepsis. When that was elevated a relatively small pocket of pus in the right lower quadrant was broken into. It probably was secondary to three fistulas in that region. In lifting the transverse colon the examiner broke into a very large, greenish-black abscess, which involved the region of the hilus of the spleen and tail of the pancreas, and extended up under the left diaphragm, which corresponds to gas seen on the x-ray film. Surrounding this abscess and all over the omentum close by was a lot of chalky-white material characteristic of fat necrosis. Further dissection showed that the entire tail of the pancreas had become necrotic and that the pancreatic abscess had extended medially to involve the upper surface of the body and head of the pancreas. I should think that the entire process was probably due to a pancreatitis that perhaps started slowly and finally produced an overwhelming infection of the pancreas. The abscess had broken into the transverse colon, and that undoubtedly added to the sepsis and accounted for some of the gas that we saw on the film. There was no primary lesion in the stomach or any part of the colon. The small-bowel loops were matted together in the right lower quadrant, but only there. I do not understand the description, at the first operation in the other hospital, of the purplish bowel — unless some loop was strangulated at one time and then untwisted. We did not find anything like that, except the sepsis between the loops of bowel in the right lower quadrant.

Dr. Bartlett: Did you find any cause for the small-bowel fistula?

Dr. Castleman: Nothing except the sepsis around the colostomy. He had a large, fatty liver (weighing 2700 gm.), which might have been expected in a chronic alcoholic. The spleen was also enlarged (weighing 400 gm.), but that may have been due, in part, to the sepsis. There was no fluid in either pleural cavity. Both lungs were pushed superiorly by the elevated diaphragm, and, in addition to the atelectasis, there was moderate pulmonary edema, but no foci of pneumonia.

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## MEDICAL PREPAREDNESS

Ten years ago there was much talk of medical preparedness, along with other kinds of preparedness for war. Activity that began at that time under the banner of preparedness has been almost continuous and has often been carried on by the same individuals or committees, but the word on the banner changed, first to *defense*, then to *victory* and then to *post-war planning*. Planning for what? Why, for preparedness, of course. So now it is beginning all over again; the world changes, but not the emotions of mankind!

In a recent Washington Report on the Medical Sciences it is stated that the "Army medical department is industriously gathering information on establishment and operation of its own medical school." The statement makes it clear that nothing beyond the possible release of a few trial balloons is contemplated for the immediate present, but the idea of an Army medical school, as a means to an end, is worthy of careful consideration. In its favor would be the fact that once established, it would at least provide a group of men who had committed themselves to a career in military medicine. Although the number of such men could never provide sufficient personnel for wartime needs, it might well supply a nucleus round which civilian physicians would gather in times of emergency. It might also lead to an eventual recognition of the importance of military medicine among medical educators as a whole. On the other hand the Army's present plan of assigning a medical officer to the faculty of existing medical schools on a co-operative basis might be developed to give quicker and larger results, and at a lower unit cost to the Government.

All medical schools are today in a difficult financial position. For even the richest of universities the establishment of a new medical school is a major undertaking. An Army medical school would not be embarrassed regarding finances, however; even were it organized on a lavish scale it would be a trivial item on the federal budget. An Army school would be more apt to encounter difficulties in mustering an outstanding staff of teachers; such organization takes time, and in the matter of preparedness time again is of the essence. Unless it can be clearly seen that advantages will quickly flow, the project should not be advised.

Assuming that an adequate staff could be mustered within a period of two years and that it could be completely protected from political and other pressures (a *sine qua non* for success), it would be 1955 before the first class would emerge into anything useful from a medical point of view. An alumni body could not aggregate into useful volume before 1960 at the earliest, and the accumulation of that intangible thing called tradition, without which no institution can attain its maximum effectiveness, would lie still farther in the future.

It may be that the need is sufficiently urgent to consider initiating an Army medical school—

not to meet the present call for preparedness, but to prepare for the next time preparation may be called for. If there is reason to believe that the demand for military doctors is a long-range one that must be built into the national economy from this time forward, action should be taken. One cannot help feeling that the Army's medical record is a proud one as it stands, from Shippen to Bliss. Under these men and those who have served between them the Army has produced its Warrens, its Beaumonts and its Reeds. Could it have done so if it had been just another medical school? As one looks back over history one cannot help observing that the Medical Department of the Army has been conspicuously successful in extracting the best available talent from the profession as a whole when it was needed and in as sufficient volume as can ever be permitted by the exigencies of war.

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## TEACHING IN VETERANS ADMINISTRATION HOSPITALS

THE report on the practice and teaching of surgery in Veterans Administration hospitals, which appears elsewhere in this issue of the *Journal*, deserves careful reading and thoughtful consideration. There are several aspects of this matter that are closely tied with the socioeconomic problems of medicine today.

The execution of the plans formulated under General Hawley's tenure of office was, in large measure, the result of far-seeing policies envisioned by the late Dr. Elliott C. Cutler. The first and foremost goal was, and still is, the optimum care for the veterans in the Veterans Administration hospitals. That this primary purpose has, to a very gratifying extent, been accomplished is apparent from the report, and is clear and unmistakable to those who are associated with these "affiliated hospitals." To accomplish this, the most important step was to bring the hospitals in close connection with, and under the supervision of, the dean's committee of the various medical schools with which these hospitals are now affiliated; these dean's committees have, practically speaking, complete control of the appointment of the professional staff that serves the hospitals. The consultants, the attending surgeons and the resident staff must all be approved by the dean's committee before their names are sent to the administrative side for formal appointment. Thus, it has been possible to obtain a staff of well trained men of recognized surgical ability and with experience and interest in teaching. The selection of these men is now practically free from any political influence however well-intentioned that might be. The positions are given for professional merit alone. To quote the article, "In a word, they bring the medical school to the hospital." The senior consultants are men of known reputation as surgeons and teachers. The attending surgeons are younger, but, being free from administrative responsibilities, they do the bulk of the daily bedside and operating-room instruction. They handle the more difficult cases themselves, but, in general, their function in the operating room is to supervise and to assist personally the resident staff. To quote further, "It may be well to emphasize that the attending surgeon cannot be a man of limited experience hoping to broaden himself by operating in the Veterans Administration hospitals. He must be an accomplished surgeon, capable and willing to teach. His role is a comparatively selfless one." The presence of a well trained resident staff is a great asset to any hospital. Its advantages are obvious, and as the author of the article points out, the comparison between the hospital with the medical-school affiliation and that without one is most striking. That the care received by the patients in the affiliated hospitals is better admits of no argument. By proper utilization of the affiliated hospitals as teaching centers, great benefit comes to the patient. This policy has also provided a larger field for the proper training of the young surgeon. Both parties have benefited greatly.

What of the future? As long as the present policy continues, the patient in the Veterans Administration hospital will continue to receive a very high quality of care — as he should. But, as the author points out, such a system cannot be expanded too far without becoming administratively top-heavy, with disastrous results. "The machine would control the men, and the control of the patient would be in the grip of an automaton." But whatever

the changes that lie ahead of us in the future, the medical profession cannot allow, through inertia and lack of interest, the actual professional care of patients in Veterans Administration hospitals to drop to the low levels that were once the rule.

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## MERRY WIDOW

GREER reports on another page in this issue of the *Journal* his experiences with victims of the sable-garbed widow whose kiss is sometimes death. They are of more than passing interest, for this mariticidal vagrant, her egg sac flung over her back, her symbolic hourglass, its sands of time forever running out, stamped upon her ventral aspect, is steadily widening her range even into those austere regions of New England north of Boston.

*Latrodectus mactans*, despite her reputation, is neither sadistically merry nor brutally aggressive. Instead she is apparently a shy, timid and confused creature who kills her mate at the call of instinct rather than from sheer wantonness. She is more to be pitied than censured, for she seems doomed never to know the tranquil joys of domesticity. In view of the extreme toxicity of the venom with which she is armed, however, it is comforting to know that she attacks only when cornered, and to learn the effectiveness of calcium gluconate as an antidote for her bite.

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## "NOTES FROM THE MEDICAL EXAMINER"

IN THIS issue of the *Journal* a new department is initiated under the title "Notes from the Medical Examiner." It is the hope of the editors that this department, appearing at approximately monthly intervals, will meet with the approval of its readers and may be continued as long as it proves to be of value.

"Notes from the Medical Examiner" will be a department devoted to ground common to the specialist in legal medicine and to the clinician, whether specialist or general practitioner. It will include such matters as procedures and technical methods evolved from the study of cases terminating in the jurisdiction of the medical examiner's office; discussions of groups of cases ending fatally as a result of unpredictable deficiencies or hazards in treatment or diagnosis; discussions of the legal and medical aspects of obscure and sudden deaths; and reviews of legal medical subjects, including toxicology, pathology and clinical subjects that are now dispersed in a variety of medical and other technical journals.

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# MASSACHUSETTS MEDICAL SOCIETY

## VETERANS IN FOREIGN MEDICAL SCHOOLS

At the meeting of the Council held on October 6 a resolution (as printed in the December 2 issue of the *Journal*) disapproving a policy of the Veterans Administration advising veterans that they may attend foreign medical schools at the expense of the Government was unanimously adopted by the Council.

Copies of the resolution were sent to every Massachusetts senator and congressman. Each has received the following letter from the Veterans Administration concerning the problem:

Honorable Richard B. Wigglesworth
United States Representative
Milton, Massachusetts

Dear Mr. Wigglesworth:

This will acknowledge receipt of your letter of October 30, 1948, with which you enclosed a copy of a resolution adopted by the Council of the Massachusetts Medical Society relative to the approval by the Veterans Administration of medical schools located in foreign countries to provide training to American veterans of World War II, under the provisions of Public Law 346, 78th Congress, as amended.

The resolution has been read carefully and the contents thereof noted with interest. The statement contained in the resolution that " . . . the Veterans Administration in Washington is advising veterans that they may attend any medical school in Europe at the expense of the Government; and . . . ," is not consistent with the facts.

A copy of the *New England Journal of Medicine* was forwarded to the Veterans Administration by Dr. George C. Shattuck . . . under date of July 29, 1948. Special attention was invited at that time to the editorial "Of Thee I Sing" and the reprint of a letter addressed to the editor by Dr. A. H. A. Campbell, 520 Commonwealth Avenue, Boston 15, Massachusetts, relative to the article by Dr. J. L. Lochner, Jr., Secretary of the Board of Registration for the State of New York, which appeared in the May 1, 1948, issue of the *Journal of the American Medical Association* entitled, "Licensure Evaluation of European Medical Graduates."

Dr. Shattuck was advised under date of August 24, 1948 that Public Law 346, 78th Congress, as amended, provides in part that, "Such person (an eligible veteran) shall be eligible for and entitled to such course of education or training, full time or the equivalent thereof in part-time training, as he may elect, and at any approved educational or training institution at which he chooses to enroll, whether or not located in the State in which he resides, which will accept or retain him as a student

or trainee in any field or branch of knowledge which such institution finds him qualified to undertake or pursue: ...." (Parenthetical statement added.)

It was further emphasized in this connection that the Veterans Administration approved educational institutions to provide training under the above legislation. It does not accredit institutions, evaluate credits, or pass on the standards of one institution as compared with another. Every effort is made to approve only those institutions which are found to be qualified in accordance with the criteria established for approving educational institutions by the Veterans Administration. These criteria cover such factors as control, staff, plant and equipment, curriculum, finances, stability etc. Only in very exceptional cases are foreign educational institutions now approved which have been established subsequent to 1939.

With regard to the statement contained in the resolution that the Veterans Administration is advising veterans that they may attend any medical school in Europe, your constituents may be advised that it is the policy of the Veterans Administration to inform all veterans making inquiry concerning the possibility of pursuing medical training outside of the United States that most of the state boards of medical examiners, as well as the American Medical Association, have definite restrictions on recognition of work done in schools of medicine in foreign countries. They are informed that in more than a third of the states graduates of medical schools outside of the United States and Canada are not eligible for licensure. They are furnished with the list of eighteen states in which credentials from medical schools in foreign countries are not accepted, as shown in the *Journal of the American Medical Association*, May 13, 1945. Each inquiring veteran is informed that if he is planning to practice medicine in the United States, it is strongly advised that he check with the state board of medical examiners in the state in which he expects to practice, as to the acceptability of work from the foreign medical school which he has under consideration.

The Veterans Administration has had considerable correspondence with the Secretary of the American Medical Association relative to the matter of approval of foreign medical schools to provide training under Public Law 346. The Secretary recently furnished this office with several copies of a reprint from the *Journal of the American Medical Association*, May 17, 1947, Vol. 134, P. 278, "Requirements for Candidates for Medical Licensure on the Basis of Credentials Obtained in Countries Other Than the United States and Canada," and several copies of a directory of "Medical Examining and Licensing Boards" in the United States. Copies of this material have been furnished to each of the five offices of Attaches for Veterans Affairs located at the American Embassies, London, England; Paris, France; Rome, Italy; Mexico City, Mexico; and the American Consulate, Geneva, Switzerland. The Office of Attache for Veterans Affairs at London, has jurisdiction over Vocational Rehabilitation and Education matters in the United Kingdom, Eire, Norway, Sweden, and Denmark; the Attache at Paris has jurisdiction over France and Belgium; while the other Attaches have jurisdiction over such matters in the countries where they are located.

It may be of interest to your constituents to know that as of April 30, 1948, there were only 261 United States Veterans pursuing courses in the various fields of medicine in foreign countries under Public Law 346. Many of these veterans were pursuing postgraduate study in various specialized fields, having already obtained M.D. degrees. Fifty-five of the 261 were enrolled in approved schools in Canada. Forty-two of the number were pursuing training in England and Scotland. Four were enrolled in Ireland.

In conclusion it may be emphasized that it is not necessary for a veteran to inform the Veterans Administration that he intends to practice his profession in the United States or in a foreign country. In every instance where inquiry has been made of this office, the veteran has been alerted to the potential difficulties he must expect in securing license to practice in the United States. Therefore, in securing a medical education in a foreign country, veterans who consult us first do so of their own volition, and with full knowledge of the obstacles which may make it impossible to practice their chosen profession in this country.

Please be assured that your interest in the welfare of veterans is very much appreciated.

O. W. Clark
*Executive Assistant Administrator*
Veterans Administration

Under Public Law 346 the Veterans Administration, by implication, is constituted the approving authority for schools of all types. Since the Veterans Administration, according to Mr. Clark's letter, has consulted the American Medical Association "relative to" foreign medical schools, it is to be hoped that the approval of that organization, which is the acknowledged authority on medical schools, will be made a *sine qua non* of recognition of any medical institution by the Veterans Administration.

If Public Law 346 contains no criterion of minimal professional standards for courses in which veterans participate and if there is no stipulation regarding the value to the veteran of the knowledge and skill acquired, the law should be amended. It is questionable whether veterans should be permitted to take courses, at the taxpayers' expense, that will be worthless. When such a course is "chicken sexing," as pointed out in the editorial "Of Thee I Sing," a waste of public money is all that is at stake, but when study in unapproved medical schools is involved, the threat to acceptable medical standards in the United States is a matter of the gravest concern to physicians.

H. Quimby Gallupe, *Secretary*

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## NOTES FROM THE MEDICAL EXAMINER

### THE TOXICOLOGY OF ALCOHOL

No chemical known to man is responsible for so many deaths, lost manpower days and traumatic injuries as ethyl alcohol. It is directly or indirectly responsible for more than 50 per cent of all deaths caused by chemical agents.[1]

*Metabolism*

Ethyl alcohol is completely absorbed from the gastrointestinal tract within one to three hours of ingestion. Its concentration after complete absorption is proportional to the water content of the individual tissue. Thus, the blood level provides a reliable index of that in brain tissue at all times in the postabsorptive period. The disappearance of alcohol from the body results from both oxidation and excretion (about 95 per cent by the former). Regardless of the dose, alcohol does not remain in the living body longer than twenty-four hours.

The oxidation probably does not exceed 5 to 10 gm. an hour or from 0.02-0.04 per cent an hour. The toxicity is purely that of an acute central-nervous-system depressant. A late manifestation of alcohol intoxication is a shift of water from an

intracellular to an extracellular situation,[2] a possible explanation for the thirst occurring during and after the alcoholic debauch.

### *Alcohol as a Cause of Death*

Alcohol may cause death either by acute narcosis or by initiating or accentuating physical and mental abnormalities in the patient and thus predisposing him to a variety of fatal misadventures.[3]

*Deaths due directly to alcoholism.* Ethyl alcohol is an anesthetic agent and in sufficient amounts kills by medullary paralysis. When death occurs relatively early in the postabsorptive phase (not the usual time for death) the blood alcohol concentration may be as high as 0.5 per cent or even higher. Frequently, death does not occur until several hours or longer after the onset of coma. Owing to continued metabolism, the post-mortem blood alcohol concentration may be well below 0.5 per cent. Alcoholic addicts frequently, by accident or design, take large doses of a barbiturate compound. There is an additive depressant action by these two anesthetic drugs.[4] Complications of the alcoholic coma may be responsible for death, such as acute asphyxiation by food aspirated into the air passages or hemorrhagic pulmonary edema from the inhalation of liquid gastric juice. A series of alcoholic debauches may render the patient more susceptible to infection, particularly of a respiratory nature.

Hepatotoxic chemicals such as the halogenated hydrocarbons may be accentuated by acute and chronic alcoholism. The evidence to date does not specifically incriminate alcohol in the pathogenesis of liver cirrhosis except upon the basis of nutritional deficiency.[5] Acute and chronic pancreatitis is said to be more common among alcoholic addicts,[6] but this has not been our experience. The pathological nervous-system change in chronic alcoholism is obscure except in relation to deficiency diseases — for example, Wernicke's disease[7, 8] and polyneuritis. Fatal circulatory collapse may develop unpredictably and uncontrollably in acute delirium tremens related perhaps to the severe physical overexertion. In several cases severe hyperthermia of undetermined cause has been observed as a terminal event.

*Deaths due indirectly to acute alcoholism.* Predisposition to misadventure accounts for more deaths than the direct pharmacologic effect. Fully 50 per cent of drivers and nearly one third of pedestrians involved in fatal accidents are termed to have been "under the influence."[9] It is not unusual for the inebriate to commit suicide — an act that he would probably not have consummated in his alcohol-free moments. About 50 per cent of persons homicidally slain had been drinking, according to the case records of the Department of Legal Medicine of Harvard Medical School. Obviously, it may be of great legal importance to know whether the perpetrator or victim of violence was under the influence of alcohol and the degree of that influence.

### *Levels of Intoxication in Relation to Blood Alcohol Concentration*

Most people show definite and obvious signs of intoxication at 0.25 per cent, a level requiring in the average adult the ingestion of approximately 12 ounces of whisky or the equivalent within a few hours. Although many persons show evidence of intoxication at lower blood levels, few, if any, fail to have obvious impairment of physical and mental faculties at concentrations higher than 0.25 per cent. High dilution of the alcohol beverage or food in the stomach may unpredictably lower the absorption rate. Those accustomed to drinking often show less severe outward manifestations of ingestion than the occasional drinker or the neophyte. Coma approximating surgical anesthesia is reached usually at levels of 0.4 per cent or above.

The National Safety Council has adopted the concentration 0.15 per cent alcohol in the blood as prima facie evidence of intoxication with respect to the operation of a motor vehicle. At this concentration not all people (perhaps 50 per cent) show significant behavior alterations on casual examination. In Evanston, Illinois, drivers with more than 0.15 per cent alcohol in the blood were fifty-five times more likely to become involved in a personal-injury accident than drivers with no alcohol.[9]

### *Specificity*

With practically all present-day methods, alcohol is determined indirectly by estimation of the volatile reducing material of the specimen by distillation or by desiccation.[10] In the presence of putrefaction, analysis is unreliable because of the formation of incompletely identified volatile reducing substances. Since liver tissue oxidizes alcohol after death, it should not be used.

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Walter W. Jetter, M.D.
*Assistant Professor of Legal Medicine*
Harvard Medical School

## MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

### COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1948

RÉSUMÉ

| DISEASE | NOVEMBER 1948 | NOVEMBER 1947 | SEVEN-YEAR MEDIAN |
|---|---|---|---|
| Chancroid | 5 | 5 | 2* |
| Chicken pox | 1740 | 753 | 969 |
| Diphtheria | 37 | 18 | 18 |
| Dog bite | 806 | 620 | 620 |
| Dysentery, bacillary | 3 | 21 | 22 |
| German measles | 59 | 60 | 60 |
| Gonorrhea | 262 | 234 | 378 |
| Granuloma inguinale | 0 | 0 | 0* |
| Lymphogranuloma venereum | 1 | 0 | 0* |
| Malaria | 1 | 5 | 15 |
| Measles | 2789 | 153 | 657 |
| Meningitis, meningococcal | 3 | 4 | 12 |
| Meningitis, Pfeiffer-bacillus | 4 | 3 | 5 |
| Meningitis, pneumococcal | 4 | 3 | 4† |
| Meningitis, staphylococcal | 0 | 0 | 0† |
| Meningitis, streptococcal | 0 | 1 | 0† |
| Meningitis, other forms | 1 | 0 | 0† |
| Meningitis, undetermined | 2 | 7 | 4† |
| Mumps | 1020 | 461 | 523 |
| Pneumonia, lobar | 62 | 48 | 162 |
| Poliomyelitis | 5 | 15 | 24 |
| Salmonellosis | 3 | 8 | 7 |
| Scarlet fever | 467 | 304 | 704 |
| Syphilis | 183 | 235 | 391 |
| Tuberculosis, pulmonary | 240 | 204 | 217 |
| Tuberculosis, other forms | 19 | 10 | 12 |
| Typhoid fever | 1 | 1 | 2 |
| Undulant fever | 2 | 3 | 4 |
| Whooping cough | 255 | 647 | 647 |

* Four-year median.
†Six-year median.

COMMENT

Those diseases above the seven-year median are chicken pox, diphtheria, measles, mumps and tuberculosis all forms.

Those diseases with incidence below the seven-year median are bacillary dysentery, poliomyelitis, salmonellosis, scarlet fever, typhoid fever, undulant fever and whooping cough.

Chicken pox for the second month in succession is highest since beginning of reporting in 1916.

For the eighth month, mumps has the highest incidence since it was made reportable in 1916.

Diphtheria has the highest incidence since 1934, and measles the highest since 1925.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Anthrax was reported from: Saugus, 1; total, 1.

Diphtheria was reported from: Abington, 1; Arlington, 2; Boston, 18; Brockton, 1; Chelsea, 1; Dartmouth, 1; Framingham, 1; Gardner, 1; Lowell, 1; North Attleboro, 1; North Reading, 1; Somerville, 1; Waltham, 3; Winchester, 3; Winthrop, 1; total, 37.

Dysentery, bacillary, was reported from: Lowell, 2; Medford, 1; total, 3.

Encephalitis, infectious, was reported from: Millbury, 1; Pittsfield, 2; Worcester, 2; total, 5.

Lymphocytic choriomeningitis was reported from: Barnstable, 1; total, 1.

Malaria was reported from: Wellesley, 1; total, 1.

Meningitis, meningococcal, was reported from: Boston, 1; Needham, 1; Spencer, 1; total, 3.

Meningitis, Pfeiffer-bacillus, was reported from: Chicopee, 1; Lynn, 1; Pittsfield, 1; Saugus, 1; total, 4.

Meningitis, pneumococcal, was reported from: Boston, 1; Northbridge, 1; Springfield, 2; total, 4.

Meningitis, other forms, was reported from: Boston, 1; total, 1.

Meningitis, undetermined, was reported from: Auburn, 1; Springfield, 1; total, 2.

Poliomyelitis was reported from: Peabody, 1; Springfield, 1; Stoneham, 1; Worcester, 2; total, 5.

Salmonellosis was reported from: Brookline, 1; Medford, 1; Wakefield, 1; total, 3.

Septic sore throat was reported from: Boston, 6; Fall River, 1; Merrimac, 1; total, 8.

Trachoma was reported from: Worcester, 1; total, 1.

Trichinosis was reported from: Everett, 1; Fall River, 1; Gloucester, 1; Hingham, 1; total, 4.

Typhoid fever was reported from: Quincy, 1; total, 1.

Undulant fever was reported from: Dartmouth, 1; Plymouth, 1; total, 2.

## MISCELLANY

### SEED FOR EUROPE

Two CARE seed packages containing potential harvests of food for human beings and fodder for livestock in Europe are now available for shipment.

Thirty-one selected varieties of vegetable seeds, enough to plant a garden up to 50 by 150 feet, are contained in the package designed for family use. The other, weighing 20 pounds, holds enough hybrid field corn seed to plant 2½ acres and provide valuable feed for fattening meat animals or maintaining a high level of production in dairy cattle.

The new CARE packages are being offered for $4 each, and orders are now being received by CARE at 50 Broad Street, New York 4, New York, as well as at all CARE offices throughout the country, for guaranteed delivery in eleven European countries. Orders should be sent at the earliest possible date to ensure delivery in time for the planting seasons.

The new packages may be ordered for delivery in Austria, Belgium, Czechoslovakia, Finland, Italy, France, Greece, the Netherlands, Poland, Great Britain (England, Scotland, Wales and Northern Ireland) and the American, British and French zones of Germany and Berlin.

## CORRESPONDENCE

### PROPER USE OF HOSPITALS

*To the Editor*: I should like to invite attention to the editorial in the *Norfolk Medical News* for November, 1948, especially the following quotation:

> Numerous surveys have shown . . .
>
> 6. Failure of hospital management to limit admissions to patients requiring hospital treatment.
>
> 7. Failure of physicians to cease hospitalizing patients who might adequately be cared for at home.
>
> 8. Tendency of physicians to continue employment of unnecessary laboratory procedures and expensive treatments. . . .
>
> A concerted effort by both management and physicians will show effective result.

I have for some time been convinced that the major effort in the solution of this is right in the lap of the doctors of the hospital staff. If they fail to take the initiative before somebody else does, they will have missed a major opportunity. It seems to me that finding No. 8 presents the chief challenge. Teaching hospitals have more reason perhaps to continue present practice, but a very desirable and far reaching reform could be initiated by making students, interns and residents (and the visiting staff too) conscious that somebody has to pay the costs and that this is one of the items of medical expense about which something can be done.

Is there anything in medical socioeconomics that is more important?

ELMER S. BAGNALL, M.D., *Chairman*
Committee on Medical Economics
Massachusetts Medical Society

## BOOK REVIEWS

*Tuberculosis: A discussion of phthisiogenesis, immunology, pathologic physiology, diagnosis, and treatment.* By Francis M. Pottenger, M.A., M.D., LL.D. 8°, cloth, 597 pp., with 105 illustrations. St. Louis: The C. V. Mosby Company, 1948. $12.00.

Those who have been brought up in the field of tuberculosis have come to know what to expect from the writings of the senior Dr. Pottenger. For nearly fifty years he has been an advocate of tuberculin therapy in pulmonary tuberculosis. He has written extensively on the importance of a very meticulous physical examination, extolling especially the fine points of inspection and palpation in the diagnosis of pulmonary tuberculosis. In fact, he claims to be able to diagnose pulmonary tuberculosis by palpation long before there is activity in the lungs and before tubercle bacilli can

be demonstrated in the sputum. All this and much more has come to be known as associated with the personality of the author.

The reviewer hoped that in this new book he would find some of the newer aspects of pathology, diagnosis and treatment that belong to the 1948 era. He looked for longer discussion of tuberculous bronchitis, its implications regarding the dynamics of cavities or the effect of bronchial stenosis on the clinical course of the disease.

The author devotes considerable space to outdoor and light treatment as well as tuberculin therapy, but gives very little detailed discussion on the indications and mode of treatment with streptomycin.

The term "compression therapy" hardly has a place in a modern book on tuberculosis, and yet it is quite loosely used together with "collapse therapy." The two are certainly not synonymous any more than two other terms — phrenicectomy and phrenicotomy — that are used indiscriminately.

Since lobectomy and pneumonectomy have a place in the general scheme of tuberculosis therapy and since this type of treatment requires the greatest experience and clinical judgment, the reader has a right to expect much more guidance on this subject than the few paragraphs allotted to it in this book.

Certainly, the author is still the great scholar and the great interpreter of phthisiogenesis with its multiform pathologic lesions and the effect it has on the clinical course of the disease. His description of the neurologic manifestations of pulmonary tuberculosis is a gem and a classic.

Knowing therefore the erudition and the long experience of the author one expects an authoritative as well as an up-to-date treatise of the subject. Instead, this book is merely another edition of his old books.

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*Atlas of Plastic Surgery.* By Morton I. Berson, M.D. 4°, cloth, 304 pp., illustrated. New York: Grune and Stratton, 1948. $15.00.

There is probably no branch of surgery that can be better described in pictorial form than plastic surgery, mainly because it consists of the treatment of surface deformities. This book is divided into eight chapters. The first covers a general discussion of the various standard procedures in the plastic repair of wounds. The remaining chapters consider the operative procedures in the correction of the various deformities of the face, with the exception of Chapter VII, which deals with mammaplasties and lipectomies. The cosmetic aspect of facial malformations is described in more detail than conditions associated with faulty function. This book of 304 pages is well illustrated with numerous diagrams and photographs.

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## BOOKS RECEIVED

**The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.**

*Practical Bacteriology, Hematology, and Parasitology.* By E. R. Stitt, M.D., Ph.M., Sc.D., LL.D., graduate of the London School of Tropical Medicine; Paul W. Clough, M.D., physician-in-charge of the Diagnostic Clinic, Johns Hopkins Hospital, and assistant professor of medicine, Johns Hopkins University School of Medicine, and associate professor of medicine, University of Maryland School of Medicine; and Sara E. Branham, M.D., Ph.D., Sc.D., senior bacteriologist, National Institute of Health, professorial lecturer in preventive medicine, George Washington University School of Medicine, and chairman, Laboratory Section, American Public Health Association, 1946-1947; and contributors. Tenth edition. 8°, cloth, 991 pp., with 765 illustrations and 7 color plates. Philadelphia: The Blakiston Company, 1948. $10.00.

This standard work has been thoroughly revised to bring it up to date since the publication of the last edition. The text is well printed with a legible type on light paper. A comprehensive index concludes the volume. Certain reference tables have been printed on the back and front covers. This is a bad practice, since they will become defaced through usage. The book is recommended for all medical libraries and all persons interested in the subject.

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*Paediatrics for Nurses.* By Arthur G. Watkins, M.D., F.R. C.P., physician in children's diseases, Cardiff Royal Infirmary, and lecturer in pediatrics, Welsh National School of Medicine. 12°, cloth, 192 pp., with 22 illustrations and 9 tables. Baltimore: The Williams and Wilkins Company, 1948. $3.50.

This manual is essentially clinical in its approach and stresses medicinal therapy. Material on nursing technic and on the infectious fevers is omitted. Printed in Great Britain, the manual reflects the British point of view.

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*Victor Robinson Memorial Volume: Essays on history of medicine in honor of Victor Robinson on his sixtieth birthday, August 16, 1946.* Solomon R. Kagan, M.D., editor. 8°, cloth, 447 pp., with 38 illustrations. New York: Froben Press, Incorporated, 1948. $10.00.

This memorial volume was intended for Dr. Victor Robinson on his sixtieth birthday, but owing to unavoidable delays it was not published until after his death in January, 1947. There are thirty-eight contributions by physicians and others interested in the history of medicine, together with a number of memorial sketches. A great many of them are biographic. The following may be noted as of special interest: "Job's Disease," by David I. Macht, the noted historian of Jewish medicine; the biographic sketch of Julius Leopold Pagel, of London; the history of the *United States Pharmacopeia*, by George Urdang; and "Jewish Medical Historians," by Dr. Harry A. Savitz, of Boston. The edition is limited to 350 copies. The volume should be in all medical-history collections.

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*Successful Marriage: An authoritative guide to problems related to marriage from the beginning of sexual attraction to matrimony and the successful rearing of a family.* Edited by Morris Fishbein, M.D.; and Ernest W. Burgess, Ph.D., professor and chairman, Department of Sociology, University of Chicago. 8°, cloth, 547 pp., with 16 illustrations and 3 tables. Garden City: Doubleday and Company, 1947. $4.50.

This comprehensive work by many authors on all phases of marriage, and problems relating thereto constitutes an encyclopedia on the subject. The text is divided into five parts: preparation for marriage; marriage; conception, pregnancy and childbirth; the child in the family; and social problems of sex in marriage.

The text is well written, showing good editorship. The printing is done with good type on lightweight paper. The work is intended for prospective brides and bridegrooms and their parents, but because of certain chapters on sexual technic and the prevention of conception, the book should not be available to adolescents. The last-named chapter probably makes the book illegal in Massachusetts under present state laws.

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*Widening Horizons in Medical Education: A study of the teaching of social and environmental factors in medicine, 1945-1946.* (A report of the joint committee of the Association of American Medical Colleges and the American Association of Medical Social Workers.) 8°, cloth, 228 pp. New York: The Commonwealth Fund, 1948. $2.75.

The general aims of this study were fourfold: to gather information on present-day teaching of the social and environmental aspects of medicine in the United States and Canada, to analyze the data obtained, to evaluate the methods and technics of instruction in use, and to offer recommendations based on conclusions drawn from the study.

The Committee considers its report as an initial probing of the subject and recommends further study along special lines by professional organizations and other interested groups, and the individual medical study and evaluation of methods and technics in this phase of teaching. The

Committee is convinced that medical social teaching is undergoing rapid change and growth. The report is recommended for all medical libraries, medical schools and interested persons.

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*Control of Pain with Saddle Block and Higher Spinal Anesthesia.* Edited by J. H. Walton, M.D. 8°, paper, 52 pp., with 5 illustrations and 12 plates. Summit, New Jersey: Ciba Pharmaceutical Products, Incorporated, 1948.

This short monograph presents the technics of spinal anesthesia in obstetrics and surgery, including abdominal section, with an additional chapter on nupercaine. The color plates are well done and effectively illustrate the text. The small volume should prove interesting to obstetricians and surgeons.

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*Children's Eye Nursing.* By James H. Doggart, M.A., M.D. (Cantab.), F.R.C.S., Eng., ophthalmic surgeon, Hospital for Sick Children, Great Ormond Street, London, surgeon, Moorfields Eye Hospital, ophthalmic surgeon, St. George's Hospital, and lecturer in ophthalmology at St. George's Hospital Medical School. 12°, cloth, 144 pp., with 93 illustrations and 4 color plates. London: Henry Kimpton, 1948. 9/6 net.

This manual has been written for nurses. The material is well organized, and the text well written. The publishing is excellent. The manual should prove useful to all nurses.

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*Nature of Life: A study on muscle.* By A. Szent-Györgyi, Department of Biochemistry, University, Budapest. 8°, cloth, 91 pp., with 22 illustrations and 7 plates. New York: Academic Press, Incorporated, 1948. $3.00.

This small volume contains a series of lectures delivered by Dr. Szent-Györgyi at the University of Birmingham and the Massachusetts Institute of Technology. The lectures summarize the work done in the author's laboratory and discuss the histologic, molecular and electronic structure of muscle, myosin and action, actomyosin and the muscle fiber.

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*The Clinical Picture of Thyrotoxicosis.* By Peter McEwan, M.A., M.B., Ch.B., F.R.C.S. (Edin.). 8°, cloth, 127 pp., with 4 plates and 5 tables. Edinburgh: Oliver and Boyd, 1948. 15s. net.

This monograph on toxic goiter has been written principally for the general practitioner, and emphasis has been placed on the clinical aspects of the disease. Thirty-one case histories have been interpolated throughout the text. The plate work is good, and the publishing is excellent. The small volume should prove interesting to physicians interested in the subject. It is pleasing to witness the return of high-class publishing in Great Britain.

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*Progress in Neurology and Psychiatry: An annual review.* Volume III. Edited by E. A. Spiegel, M.D., professor and head, Department of Experimental Neurology, Temple University School of Medicine, Philadelphia. 8°, cloth, 661 pp. New York: Grune and Stratton, 1948. $10.00.

In this third volume of a standard annual, over 2800 papers have been revised. Chapters have been added on mental deficiency and criminal psychiatry. Bibliographies are appended to the various chapters. The printing and type are excellent. A lighter paper could have been used to advantage. The series is recommended for all medical libraries and as a reference work to neurologists and psychiatrists.

# NOTICES

## ANNOUNCEMENTS

Dr. Raymond Gelfman announces the removal of his office to 83 Maple Street, Springfield.

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Dr. James A. Halsted announces that his office is now at the Faulkner Hospital, Jamaica Plain, Boston.

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Dr. Paul Graves Myerson announces the removal of his office to 1093 Beacon Street, Brookline.

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## GEORGE F. BAKER CLINIC

The Committee on Diabetes, appointed by action of the Massachusetts Medical Society, has suggested that clinical exercises in diabetes be offered without charge in various hospitals for physicians. To this end the George F. Baker Clinic at the New England Deaconess Hospital offers exercises on Monday and Friday of each week. The schedules are as follows:

Monday, 8:00–9:00 a.m., Second Floor, New England Deaconess Hospital. Case Presentations in Diabetes and Surgery of the Extremities.

Friday, 8:00–8:15 a.m. Second Floor, New England Deaconess Hospital. X-Ray Pathology in Diabetes.

8:15–9:00 a.m. Classroom, George F. Baker Building. Case Problems in Treatment of Diabetes and Complications: Pregnancies, acidosis, insulin resistance and so forth.

9:00–10:30 a.m. Medical Rounds for Visiting Physicians.

9:00–10:30 a.m. Instruction in Diet and Urine Testing (for Office Nurses, Technicians or Secretaries Sent by Physicians).

10:30–11:15 a.m. Class Teaching in Diabetes. (Physicians, Office Assistants and Nurses are welcome.)

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## HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society will be held in the amphitheater of Building D, Harvard Medical School, on Tuesday, January 11 at 8:00 p.m. The chairman for the evening is Dr. Raymond D. Adams.

### Program

The Correlation between Morphologic and Functional Disorders in the Motor-Nerve Cells in Poliomyelitis. Derek E. Denny-Brown and Joseph M. Foley.

An Encephalomyelitic Virus of Mice Causing Demyelinating Lesions of the Central Nervous System. F. Sargent Cheever, O. T. Bailey and A. M. Pappenheimer.

Pathological Changes in Carbon Monoxide Poisoning. Charles S. Kubik.

Subsequent meetings will be held on February 8, March 8, April 12 and May 10.

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## MEDICAL SOCIETY OF THE STATE OF NEW YORK

The annual meeting of the Medical Society of the State of New York will be held at the Hotel Statler, Buffalo, from May 2 to 5, inclusive.

(*Notices concluded on page xix*)

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**The *Journal* lacks extra copies of the July 29 and August 5, 1948, issues. If any subscribers who do not bind their copies have the above-mentioned issues on hand, the *Journal* will gladly pay 15 cents for each copy left at or mailed to its office (8 Fenway, Boston 15).**